

## MASTER OF PHILOSOPHY

### The physiological and biomechanical changes during a 100m breaststroke swim

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# **The physiological and Biomechanical Changes During a 100 m Breaststroke Swim**

**By**

**Samuel W. Oxford**

**June 2012**



# **The physiological and Biomechanical Changes During a 100 m Breaststroke Swim**

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*A thesis submitted in partial fulfillment of the University's requirements for the  
Degree of Master of Philosophy*

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## **Dedication**

I would like to dedicate this thesis to my boys Izaac Wellesley Oxford and Theo Joseph Oxford.

## Abstract

This thesis investigated changes in kinematics, including arm-leg co-ordination, ( $n=26$ ), intra-cyclic velocity fluctuations and arm-leg co-ordination ( $n=18$ ), duration and coordination of muscle activity in the upper and lower extremity muscles using surface electromyography (sEMG) ( $n=8$ ), and evaluated fatigue in selected upper body, trunk and lower limb muscles by means of frequency parameters of sEMG ( $n=8$ ) during a maximal 100 m breaststroke swim performed from a water start.

Arm-leg co-ordination was defined using two transition phases that described the continuity between the recovery and propulsive phases of the arms and the legs: CPhase 1, corresponding to the time between the end of the leg propulsion and the start of the arm propulsion phases; and CPhase 2, corresponding to the time between the end of the arm propulsion phase and the start of leg propulsion phase. Intra-cyclic velocity fluctuations were calculated from peak velocities of the greater femoral trochanter during the arm and leg propulsive phases and the minimum velocities attained during the recovery of the arms and the legs. Video analysis (50 Hz) was used to analyse swim speed (not influenced starting, turning or finishing techniques), stroke length, stroke rate and stroke efficiency. Surface electromyography (sEMG) was collected from the pectoralis major, latissimus dorsi, posterior deltoids, biceps brachii, triceps brachii, biceps femoris, rectus femoris, vastus lateralis, vastus medialis, gluteus maximus, erector spinae and rectus abdominals.

Over the duration of a 100 m breaststroke swim there was a significant decrease in the clean swim speed ( $p < 0.05$ ) with subsequent decreases in stroke length and stroke efficiency with no significant change in stroke rate ( $p > 0.05$ ). As a consequence of a decrease in clean

swim speed there were alterations in the arm-leg co-ordination with a significant decrease ( $p < 0.05$ ) in CPhase1 of the stroke cycle. There were also significant decreases ( $p < 0.05$ ) in the active phase (AF) of the major upper and lower muscles used in breaststroke swimming. There was a no significant change ( $p > 0.05$ ) in the mean power frequency (MNF) of any of the muscles that were studied.

It was concluded that participants in the study were affected by fatigue and used compensatory mechanism and strategies in an attempt to maintain swim speed, which resulted in alterations in the alterations in the combination of SR and SL, altered intra muscular co-ordination and alterations in the arm-leg co-ordination within the stroke cycle.

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**Publications**

Oxford, S., James, R., Price, M. and Payton, C. (2008) 'Co-ordination Changes in Sprint Breaststroke Swimmers during a 100 m Short Course Swim From a Push' 16<sup>th</sup> FINA World Sports Medicine Congress. Manchester UK.

Oxford, S., James, R., Price, M. and Payton, C. (2010) 'Co-ordination changes during a maximal effort 100 m short course breaststroke swim', Biomechanics and Medicine in Swimming XI, Oslo.

Oxford, S., James, R., Price, M. and Duncan, M, (2013) 'Changes in Hand Grip and Leg Strength Following a 100-m Short Course Breaststroke Swim' UK Strength & Conditioning Conference (Poster Presentation).

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## **Abbreviations**

AP = Action potentials

ADES = Active Drag Evaluation System

ARV = Average rectified value

CM = Centre of mass

CNS = central nervous system

CSS = Clean swim speed

CV = Conduction velocity

DFT = Discrete Fourier Transform

EMG = Electromyography

EPOCH = moving time window

FFT = Fast Fourier Transform

iEMG = Integrated EMG

MAV = Mean amplitude value

MDF = Median Frequency

MPF = Mean power Frequency

MUAP = Motor unit action potentials

MVC = Maximal voluntary contraction

MVIC = Maximal voluntary isometric contraction

PSD = Power spectrum density

RMS = Root mean Squared

sEMG = Surface electromyography

SL = Stroke Length

SR = Stroke Rate

SI = Stroke Index

## **CHAPTER 1**

### **LITERATURE REVIEW**

## CHAPTER 1

### 1.0 Literature Review

### 1.1 Stroke Kinematics

**Table 1.1** The FINA regulations that govern the breaststroke technique from 2009 -2013

(FINA, 2012).

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A competitive swimmers objective is to cover the race distance in the fastest time possible within the regulations of the event (Table 1.1). The total time it takes to complete the race distance can be split into four sections; i) time spent starting; ii) time spent stroking; iii) time spent turning; iv) time spent finishing (Pai, Hay and Wilson, 1984). The time spent stroking is governed by the distance covered whilst stroking and by the average speed. Mean

clean swim speed (CSS, speed not affected by starting, turning or finishing) is determined by the average stroke length (SL, the distance (m) covered during one stroke) and the average stroke rate (SR, the number of stroke cycles per second (Hz)) (Equation 1.1).

$$\text{Equation 1.1} \quad \text{CSS} = \text{SL} \times \text{SF}$$

Clean swim speed is a fundamental variable, that varies between individuals thereby having a large effect on locomotor performance in swimming (Fritzdorf, Hibbs and Kleshnev, 2009). The combination of stroke rate and stroke length is highly individual (Swain & Reilly, 1983) as swimmers find the optimal compromise between stroke rate and stroke length at the highest mean swim speed over the whole race (Sidney et al., 2011). In front crawl swimming many authors have reported evolutions in stroke rate as swimmers progress which have been shown to alter with sex (Craig et al., 1985) and skill level (Chollet et al., 1996). In breaststroke swimming the main factor that has been shown to determine final race time is mid pool speed (Pai et al., 1984; Thompson et al., 2000; Kjendlie et al., 2006). During a race the correlation between stroke rate and stroke length deteriorates as the swimmers are losing speed as a result of the decrease in stroke length which is not compensated enough by the increase in stroke rate. It has been reported (Thompson et al., 2004) that stroke rate decreases during the second lap of a 200 m breaststroke race then increases over the remaining three lengths. This “U” shaped pattern of stroke rate changes has also been reported by Kjendlie et al., (2006) in 42 medal winners out of 50 during the 2002 European Championships. The knowledge of the breaststroke kinematic changes, are a valuable tools for the coach and the swimmer (Sidney et al., 2011). It has been shown that the decrease in stroke rate is a specific characteristic of the breaststroke. One question immediately arises from this particular characteristic of the breaststroke is what is causing this dramatic change

in terms of spatial-temporal characteristics (Sidney et al., 2011). A study by Takagi et al (2004) has provided some interesting clues about this question but has not fully answered the question. Takagi et al., (2004) reported that better swimmers had longer arm glide durations and tended to lose less speed during the non-propulsion part of the stroke. From this study it can be pointed out that elite swimmers are: 1) able to minimise their speed drop by being more streamlined during the recovery and glide phases of the stroke; 2) that the adjustments to the co-ordination pattern according to distance is a key factor for success in breaststroke swimming. However what this study does not answer is the question of what is happening to the spatial-temporal characteristics between laps with in a swim and the within race changes in stroke rate and stroke length.

Amongst the factors that influence the relationship between stroke rate and stroke length and therefore overall swim speed, anthropometric parameters appear to be of importance (Keskinen, Tilli and Komi, 1989; Pelayo et al., 1996). In general, taller and bigger swimmers can produce more work per stroke, (Fritzdorf, Hibbs and Kleshnev, 2009) and therefore their stroke length is longer. The smaller swimmer cannot achieve such long stroke lengths thus they utilise a higher stroke rate (Fritzdorf, Hibbs and Kleshnev, 2009). Therefore, stroke length and stroke rate are inversely proportional at constant speed. It has previously been shown that a difference in anthropometry between the sexes mediates differences in stroke length, stroke rate and velocity in front crawl swimming (Chatard, Lavoie and Lacour, 1991; Grimston and Hay, 1986). Male swimmers have been reported to have greater stature (height) and longer segment lengths which have also been linked to greater propelling efficiency and longer stroke lengths in front crawl swimming (Toussaint, Janssen and Kluft, 1991; Kjendlie, Stallman and Stray-Gundersen, 2004). Stroke index (SI) as defined by Costill et al., (1985) as the product of average velocity ( $v$ ) and stroke length



(SL) is considered a valid indicator of swimming efficiency. Female swimmers are reported to be more efficient than their male counterparts in breaststroke swimming due to the elevated position they adopt in the water. This is a result of better buoyancy in females due to greater percentage body fat in the thighs than males (McLean and Hinrichs, 1998) resulting in a higher hip position. The higher hip position reduces the active drag by reducing the cross sectional area exposed to the water's flow (Leblanc, Seifert and Chollet, 2010). The authors showed that floatation parameters have an impact on the gliding phase of the breaststroke cycle. In particular, in the female group and at slow pace, glide duration is correlated with hydrostatic lift ( $r = .62$ ) and with maximal glide length ( $r = .44$ ); mean glide velocity is correlated with hydrostatic lift ( $r = .73$ ). In the male group and at slow pace, the sinking force was correlated with the glide phase ( $r = -0.66$ ) and with the mean glide velocity ( $r = -0.78$ ). At sprint velocity, the hydrostatic lift is correlated with the glide phase in the female group ( $r = .52$ ). The effect of the increased buoyancy in female swimmers seems to be of less importance during sprint events as the buoyancy effect is only an advantage during a certain range of velocity (Leblanc, Seifert and Chollet, 2010). This is due to the fact that in sprint events the swimmers have a higher linear velocity which causes the legs to be raised as a result of the increased hydrostatic lift forces (Miyashita and Tsunoda, 1978; Kjendlie, Stallman and Stray-Gundersen, 2004). What is unclear is how stroke efficiency changes during a swim and whether it differs between males and females.

## **1.2 Stroke Phases**

### *1.2.1 The Arm Stroke*

There are inter-individual variations in arm stroke pattern (Maglischo 2003) however there is a typical sequence of movement patterns that are characteristic of the arm stroke

(Figure 1.1). These sequences of movement patterns for the arm stroke can be divided into four distinct phases; the outswEEP, the catch, the insweep and the release and recovery (Maglischo 2003; Figure 1.1). The outswEEP begins the arm pull phase of the stroke with the arms extended out in front of the swimmer, parallel to the surface of the water. The hands are pitched at about 45 degrees outwards from the pronated position with a slight bend at the wrist as the arms move laterally into the catch position (Figure 1.1).

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**Figure 1.1** Anterior, lateral and ventral views of the arm stroke phases (Maglischo, 2003)

The catch of the arm stroke takes place just outside of the shoulders (position 2 on Figure 1.1) by which point the hands achieve a backwards orientation. The insweep can be broken into two phases (between position 2-3 on Figure 1.1), the downswEEP section of the

insweep begins from the catch position where the arms begin to bend at the elbow and move outwards and downwards in a circular motion. During this part of the downsweep the palms are still facing laterally but are starting to turn inwards. At the end of the downsweep phase the arms are at their deepest point with a near 90 degrees bend at the elbow. At the end of the first part of the insweep the hand motion changes from downwards and outwards to an inwards and upwards motion with the pitch of the hands changing to inwards and slightly upwards this continues the circular movement of the arms (Figure 1.1). The hands pass directly beneath the elbows as they approach the midline of the body (Figure 1.1). Once the hands come together under the shoulders they start the recovery phase (position 3 on Figure 1.1). The recovery phase starts from the initial forwards movement of the hands as the arms are extended forwards parallel with the water and the head returns to the water. The major muscles that are used during the arm stroke are shown in Table 1.2.

### *1.2.2 The Leg Stroke*

The kick can be divided into five phases (Maglischo, 2003) the recovery phase, the outstroke, the catch, the insweep and lift and glide phase. The kick starts with the outstroke phase of the stroke (position 2-3 on Figure 1.2) this begins with the knees bent at an angle greater than 90 degrees. The feet are above the gluteus muscle group with the feet close to each other. The pitch of the feet is altered so that the soles of the feet are facing backwards and upwards, and the foot is turned so that the toes are pointing laterally (position 3 on Figure 1.2) and are slightly outside the line of the shoulders. This is where the catch (position 3 on Figure 1.2) of the leg kick takes place (Chollet et al., 2004) and is where swimmers start to generate propulsion (Maglischo, 2003; Takagi et al., 2004). From the catch the swimmers move the legs outwards, backwards and inwards in a semicircular pattern (position 3-4 on

Figure 1.2) through extension of the legs during this phase which results in the legs moving downwards and outwards till they reach their widest part. During this phase the pitch of the feet changes to inwards. Once the legs have finished extending there is the lift and glide phase (position 4-5 on Figure 1.4). During the lift and glide phase the legs move up into alignment with the body prior to the start of the recovery phase. The recovery phase (position 1-2 on Figure 1.2), or the preparation phase as it's sometimes referred to (Counsilman and Counsilman, 1994), of the kick is where the heels are brought up to the buttocks through the rotation of the hip and medial rotation of the thighs and flexion of the knees (position 1-2 on Figure 1.2). The major muscles used during the leg stroke phase are presented in Table 1.3.

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**Figure 1.2** Frontal, lateral and ventral views of the breaststroke leg kick (Maglischo, 2003). See main text for reference to the numbered positions

### **1.3. Inter Limb Co-ordination**

Motor co-ordination involves the combination of spatial direction of body movements (kinematic parameters) and the application of force (kinetic parameters) that create desired actions or movements. Motor co-ordination involves the sequential movement of limbs or body parts in a combined manor to produce efficient process to achieve the desired outcome. Motor co-ordination involves the integration of proprioceptive information regarding the position and movement of the musculoskeletal system with neural process in the brain and spinal cord (Haken, Kelso and Bunz, 1985). Inter-limb co-ordination is the study of how movements are co-ordinated across limbs (Kelso, 1984). The analysis of co-ordination attempts to understand the laws that govern spatial temporal pattern formation in complex tasks (Kelso, 1995; Kelso, 2008). Co-ordination dynamics capture the time evolution of a system according to laws of motion exploring how these patterns of co-ordination become stable and attractive or unstable and changing in response to environmental or task demands (Kelso, 1995; Kelso, 2008). Environmental constraints are external to the individual and can be physical, reflecting the environmental conditions of the task such as light, temperature altitude, gravity and buoyancy. In swimming the changing environment constraints relates to the changes in aquatic resistance and swimming speed as drag is related to speed squared (Seifert, Buton and Davids, 2013) . Task constrains include the rules that govern the task, instructions and or equipment (Newell, 1986). Individuals can structurally alter their motor behaviour without compromise their function. This provides evidence for adaptive and role of movement pattern variability in order to satisfy the task constrains. Practical considerations in swimming are that arm and leg movements can be effectively performed but occur without efficient arm and leg co-ordination.

Wannier, et al., (2001) demonstrated that the patterns of arm and leg co-ordination that are present in walking are similar and current in other locomotor activities such as swimming. In swimming like other cyclic activities the performer has to overcome drag and generate propulsive forces. Swimming performance is dependent on the swimmers ability to overcome higher levels of active drag, than in walking, due to the aquatic environment and to produce effective propulsive forces which provide the highest efficiency (Toussaint and Beek, 1992). Success in swimming is determined by the ability to generate propulsive force while reducing the resistance to forward motion (Chollet and Seifert, 2010). To achieve this swimmers' need to coordinate the movement of their limbs in an effective and efficient manor to achieve maximum forward body displacement (Chollet and Seifert, 2010). Effective co-ordination does not only serve to propel the body forwards but also to favour breathing actions, gliding and buoyancy (Seifert et al., 2011). Thus the inter-limb co-ordination in breaststroke swimming must be analysed regarding the task-goal.

### *1.3.1 Co-ordination of the Arms and Leg Phases in Breaststroke Swimming*

The first studies that investigated inter-limb co-ordination in human swimming were those of Nemessuri & Vaday, (1971), who investigated motor patterns in front crawl swimmers, and Nemessuri & Vaday, (1971) who investigated motor patterns in breaststroke swimmers. The authors defined co-ordination as “schemes of structural circle of breaststroke as a cyclic and synchronously symmetrical form of locomotion”.

Breaststroke swimming appears to be a complex cyclic movement, for which the challenge is to organise the inter-limb coupling in order to overcome the environmental

constraints (Toussaint and Truijens, 2005). The breaststroke swimmer has to manage the transition of above and below movements in response to the FINA rules (Table 1.1). The breaststroke swimmer also has to optimise the hydrodynamics of their body position and to minimise discontinuity in propulsion to avoid high intra-cyclic velocity variations that would both lead to increases in drag (Colman et al., 1998).

In walking and running locomotion (Cavagna and Kaneko, 1977) mechanical power of locomotion ( $\dot{W}_{tot}$ ) (equation 1.1) is the sum of the power needed to accelerate and decelerate the limbs with respect to the centre of mass (the internal power,  $\dot{W}_{int}$ ) and the power needed to overcome external forces (the external power,  $\dot{W}_{ext}$ ):

*Equation 1.1* 
$$\dot{W}_{tot} = \dot{W}_{ext} + \dot{W}_{int}$$

In aquatic locomotion the external work ( $\dot{W}_{ext}$ ) can be further explored (di Prampero, Pendergast and Zamparo, 2011) into the power to overcome drag that contributes to propulsion ( $\dot{W}_d$ ) and the power that is wasted and does not contribute to propulsion ( $\dot{W}_k$ ) as water does not allow for a solid push-off (equation 1.2). Both  $\dot{W}_d$  and  $\dot{W}_k$  give water kinetic energy but only  $\dot{W}_d$  contributes to propulsion (Daniel, 1991).

*Equation 1.2* 
$$\dot{W}_{ext} = \dot{W}_d + \dot{W}_k$$

The efficiency with which the overall mechanical power is converted into useful mechanical power by the swimmer is termed propelling efficiency ( $\eta_p$ ). The relationship between the external mechanical power and its conversion into useful mechanical power is termed the Froude efficiency ( $\eta_F$ ) (equation 1.3). The Froude efficiency does not take into account the metabolic expenditure thus the efficiency with which the metabolic power input ( $\dot{E}$ ) is

converted into useful power output this is termed performance or drag efficiency ( $\eta_D$ ) (equation 1.4) (di Prampero, Pendergast and Zamparo, 2011). In swimming efficiency is discussed as gross or mechanical efficiency ( $\eta_O$ ) (Toussaint et al., 1988; Toussaint, Janssen and Kluft, 1991; Zamparo, 2006; Zamparo et al., 2008) which is the efficiency with which the metabolic power input ( $\dot{E}$ ) is transformed into useful mechanical power output (equation 1.5)

*Equation 1.3* 
$$\eta_F = W_d / W_d + W_k$$

*Equation 1.4* 
$$\eta_D = \dot{W}_d / \dot{E}$$

*Equation 1.5* 
$$\eta_O = \dot{W}_{tot} / \dot{E}.$$

Movement in swimming is an energetically costly activity with values of  $\eta_D$  ranging from 0.03 -0.09 (di Prampero et al., 1974; Holmer, 1972; Pendergast et al., 1977; Toussaint et al., 1988) indicating that less than 10% of the mechanical power output can be transformed into useful mechanical power to overcome drag forces. The better the technical ability of a swimmer the higher and more stable the clean swim speed will be for the same amount of energy release, thus reducing the intra-cyclic velocity variations. However there are no studies that have thus far reported intra-cyclic velocity variations between laps of a breaststroke swim.



More than any other stroke the breaststroke undergoes variations in intra-cyclic velocity because of the larger resistance experienced during the recovery phases of the arms and legs whilst underwater (Craig, Boomer and Gibbons, 1979; Maglischo, Maglischo and Santos, 1987). As a consequence of high resistive drag during the recovery phases it is not recommended to recover the arms or legs in isolation (Leblanc, Seifert and Chollet, 2009). The expert breaststroker's co-ordination is characterised by synchronised recovery times which results in the propulsion of one set of limbs when the other set are in a hydrodynamically optimised position (Chollet et al., 2004; Seifert and Chollet, 2005; Takagi et al., 2004).

In breaststroke swimming the timing of the arms and legs is a key concern with regards to performance. There is a significant relationship between upper and lower limb co-ordination and overall swim velocity (Chollet et al., 2004; Chollet, Tourny-Chollet and Geizes, 1999). The breaststroke, stroke cycle is composed of three main phases of the legs and three main phases of the arms (Figure 1.1 and 1.2) These are the propulsive phase, recovery phase and the glide phase (Leblanc, Seifert and Chollet, 2009). Depending on the time spent in the glide phase, three main types of overall breaststroke co-ordination have been identified: 1) *Glide* co-ordination is characterised by a glide phase following the end of the leg kick, prior to the start of the arm stroke; 2) *Overlap* co-ordination is characterised by the initiation of the arm phase before the finish of the leg kick; 3) *Continuous* co-ordination is characterised by the start of the arm phase immediately following the completion of the leg kick (Chollet, Tourny-Chollet and Geizes, 1999; Leblanc, Seifert and Chollet, 2009; Maglischo, 2003).

Research suggests that the different co-ordination patterns tend to be associated with the different race distances (50 – 200 m) used in competitive breaststroke swimming. The 200 m event is associated with the glide co-ordination pattern, 100 m with the continuous co-ordination pattern, and the 50 m with the overlap co-ordination pattern (Chollet, Tourny-Chollet and Geizes, 1999; Leblanc, Seifert and Chollet, 2009; Maglischo, 2003). As race distance decreases from 200 – 50 m the timing of the arms and legs phases of the stroke change. The time gap between the arm and leg propulsive phases has been termed the arm-leg-lag phase of the stroke (Leblanc et al., 2005; Takagi et al., 2004). This arm-leg-lag phase of the stroke has been reported to be positive (glide co-ordination) or negative (overlap co-ordination). This area of research has been given some attention in previous years (Chollet et al., 2004; Leblanc et al., 2005; Leblanc, Seifert and Chollet, 2009; Seifert and Chollet, 2005) as it has been reported that there is a need to understand this phenomenon.

The majority of the research that has looked at the time gap between the arms and the legs has focused on intermittent protocols of 25 m swims at paces associated with 50, 100 and 200 m performance times (Chollet et al., 2004; Leblanc et al., 2005; Leblanc, Seifert and Chollet, 2009; Seifert and Chollet, 2005). The major findings of all these studies have been that as race pace decreases there is a decrease in the arm-leg-lag phase of the stroke. This shift in co-ordination is a means for the swimmers to increase the propulsion phase of the stroke.

Inter-limb co-ordination has been quantified from the use of video analysis systems that are comprised of a minimum of three cameras with a minimum recording capacity of 50Hz (Chollet and Seifert, 2010). One camera is positioned in the frontal view, one providing

sagittal view below the water and one the sagittal view above the water' The cameras are genlocked together to allow video synchronisation.

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**Figure 1.3** Arm to leg co-ordination in breaststroke (Seifert and Chollet, 2005).

In the study of co-ordination of the breaststroke, the arm phase (Figure 1.1) has been divided into four or five phases depending on the researchers (Chollet et al. 2004; Chollet & Seifert 2008; Leblanc et al. 2005; Takagi et al. 2004) (Figure 1.3). The four phases of the arm stroke are: arm-glide, elbow push, first part of recovery until an arm / forearm angle of  $90^{\circ}$ , second part of the recovery (Chollet et al., 2004; Leblanc et al., 2005; Leblanc, Seifert and Chollet, 2009; Seifert and Chollet, 2005). The leg stroke is also composed of five phases (Figure 1.4): leg propulsion; leg insweep; leg glide; the recovery is split into two phases of which the first part is until a thigh leg angle of  $90^{\circ}$  (Chollet et al., 2004; Leblanc et al., 2005;

Leblanc, Seifert and Chollet, 2009; Seifert and Chollet, 2005). As a result of the utilisation of these arm and leg phases it has been possible to identify five time gaps within the stroke cycle for characterising the time between arm and legs (Chollet et al., 2004; Leblanc et al., 2005; Leblanc, Seifert and Chollet, 2009; Seifert and Chollet, 2005). These time gaps allowed the determination of the arm-leg co-ordination (Chollet et al., 2004; Leblanc et al., 2005; Leblanc, Seifert and Chollet, 2009; Seifert and Chollet, 2005). The time gaps were:

**T1<sub>a</sub>**, was characterised as the time between the end of the leg propulsion and the start of the arm propulsion

**T1<sub>b</sub>**, was characterised as the time between the end of the leg insweep and the beginning of arm propulsion

**T2**, was characterised as the time between the start of the arm recovery and the start of the leg recovery

**T3**, was characterised as the time between the end of arm recovery and the end of leg recovery

**T4**, was characterised as the time between 90° of arm flexion in arm recovery and 90° of leg flexion in leg recovery

**TTG**, was the sum of the absolute values of the four time gaps and was expressed as a percentage of the total stroke.

The work of Takagi, et al., (2004) divided the arm stroke into four phases which were 1) recovery phase, which corresponded to the time period from the maximal flexion of the elbows underneath the breast until the arms were completely stretched in front of the face; 2) glide phase, which corresponded to the time from when the arms were completely stretched forward until the first observed lateral movement of the hands; 3) outswEEP, the time from the

end of the glide-phase to the first observed down and backwards movement of the hands; 4) insweep phase, the time from the end of the outswEEP phase to the hands coming together for the recovery phase. Takagi, et al., (2004) classified the outswEEP and the insweep for propulsive phases in accordance to Schleihauf, et al., (1988) and Thayer, et al., (1986) who reported that there is a possibility of propulsion from the outswEEP phase of stroke.

Takagi, et al., (2004) separated the leg stroke into three phases which were defined as: i) sweep phase, the time from maximum dorsi flexion of the ankle to maximum extension of the knees as the feet came together; ii) lift and glide, the time from the end of the sweep phase until the legs are in line with the body just beneath the surface; iii) recovery phase, the time from the first observed forward movement of the feet by flexing at the knees to maximum flexion of the knees. Takagi, et al., (2004) reported that during the leg kick the only propulsive phase was the sweep phase.

To allow the analysis of arm-leg co-ordination, Takagi, et al., (2004) identified three key phases of the stroke cycle and expressed them as a percentage of the stroke cycle, the phases were: i) *simultaneous recovery time*, which was an indicator of the non-propulsive phase of the stroke, the difference between the end of the arm propulsion phase and the start of the legs sweep; ii) *Arm lag time*, which was the time from the start of the leg propulsion to the beginning of the arm propulsion divided by stroke duration; iii) *simultaneous propulsion time*, which indicated simultaneous propulsion of the arm and legs calculated as the time difference between the end of the leg sweep and the start of the arm outswEEP.

The research regarding arm-leg co-ordination has shown that as race pace increases from 200 to 50 m the glide phase has been shown to be reduced in the stroke cycle. This

adaptation of the arm-leg co-ordination showed that a higher propulsive continuity between the upper and lower limbs propulsion mostly related to the glide time changes rather than the synchronisation of the arm and leg recoveries (Chollet et al., 2004; Leblanc et al., 2005; Seifert and Chollet, 2005). This view was supported by Takagi, et al., (2004) who state that arm and leg propulsion have a common effect on the propulsion of the whole body. The research of (Seifert and Chollet, 2009) has shown relationships amongst speed, stroke rate, stroke length and co-ordination using polynomial regression. It was reported that stroke rate, stroke length and speed may influence co-ordination suggesting that coaches and scientists could use the glide phase to identify an individual's best race distance and to distinguish the time gaps that are most effective for performance at specific race distances. Co-ordination shows differences with sex and skill (Leblanc et al., 2005; Leblanc et al., 2007; Leblanc, Seifert and Chollet, 2009) in breaststroke swimming. Fatigue has also been shown to alter the timing of the arm phases of the stroke in front crawl swimming (Toussaint, et al., 2006), to date there are no studies that have investigated the effects of fatigue in breaststroke swimming. There is a need to investigate the fatigue effects in breaststroke swimming as the findings from front crawl cannot be related to that of breaststroke swimming due to the characteristic differences in the way that swimmers use of their arms and legs in breaststroke compared to that of front crawl. A limitation of the previous research in breaststroke is that there are no investigations to the authors knowledge that have investigated the inter lap changes in stroke kinematics and co-ordination over a simulated race distance. All previous research has investigated changes at similar stages of a swim (Takagi et al., 2004) or have used discontinuous protocols (Chollet et al., 2004; Leblanc et al., 2005).

## **1.4 Linear Velocity Fluctuations**

The breaststroke is the slowest of the four competitive strokes due to the large fluctuations that occur in the linear velocity during each stroke cycle. Swimmers generate large forces during the propulsive phases of the arm and leg phases. There is also considerable deceleration during the recovery of the legs in preparation for the leg kick (Maglischo, 2003). In the other three competitive strokes swimmers only normally experience a decrease of about a third in their linear velocity, whereas breaststrokers can almost come to a complete stop (Maglischo, 2003). These large changes mean that breaststroke swimmers must apply large forces to accelerate back up to race speed (Maglischo, 2003).

The different styles of breaststroke have repercussions on the intra-cyclic velocity variations within the stroke cycle. It has been reported that during the propulsive phases of the undulating style there is a reduced amount of acceleration generated from the arms and legs, however, undulating style is also characterised by reduced amounts of deceleration during the recovery phases of the legs and arms (Colman et al., 1998; Van Tilborgh, Eustache and Persyn, 1988). During the arm and leg recovery phases, the velocity loss is around 17 % for the undulating style compared to 27 % for the flat style (Seifert et al., 2010). These differences in velocity variation have been attributed to the adopted body position during the leg propulsion phase in the undulating technique which has been reported to be less favourable (Persyn, et al., 2005). Persyn, et al., (2005), report that this body position results in the legs being pushed downwards and backwards whereas the arms and trunk plunge forwards. The body position at the completion of the arm propulsion phase in the undulating

style is characterised by a cambered back (Persyn and Colman 2005) which has been reported to increase drag forces (Colman et al., 1998).

#### *1.4.1 Hip and Centre of Mass Kinematics*

Tracking the movements of the hip or centre of mass (CM) are considered ways to analyse the body's kinematics, however, it has been shown that the hip it is not a valid estimate of the centre of mass during swimming (Barbosa et al., 2003; Mason, Trong and Richards, 1992; Psycharakis and Sanders, 2009; Figueiredo et al., 2009). The hip intra-cyclic velocity presents greater variations than the centre of mass. However, the use of the hip has been shown to be a reliable assessment of intra-cyclic velocity with a reliability of  $r = 0.95$  between the hip and the centre of mass (Costill, Lee and D'Acquisto, 1987). Which has been supported with the findings of (Capitao et al., 2006) who confirmed the correlation of  $r = 0.89 - 0.96$  in breaststroke swimmers. However, movement of the hip does not represent the changes in centre of mass due to changes in inter limb action during a stroke cycle. Although usage of the hip causes a bias, such assessment of anatomical landmarks is still used by some research groups. This is due to the fact that it allows the assessment of linear velocity over a longer distance up to 50 m which allows the analysis of cycle to cycle variability (Barbosa et al., 2011) while 3D video analysis only analyses one cycle. The velocity meter system also has the benefit of providing real time feedback to the coaches and the swimmers which is not available in the 3D video systems.

The most common assessment that is related to the hip and/or centre of mass is the assessment of the intra-cyclic variation of horizontal velocity. Throughout the stroke cycle the kinematics of the body is not uniform (Barbosa et al., 2011) as there will be increases and



decreases in the body's velocity due to movements and actions of the body's limbs. The assessment of horizontal velocity has been proposed as one of the most important biomechanical variables to be determined in competitive swimming (Kolmogorov and Duplischeva, 1992).

The velocity-time curves of the hip are shifted by 3-5 % earlier than the centre of mass, and the hip attains peak velocity values 20 % and 5 % higher than the CM during the propulsive phases of the arms and legs, respectively, with only a 1 % difference in velocity during the arm-leg phase (Leblanc et al., 2007). The coefficients of correlation between the two displacement curves have been found to be significant and to follow similar patterns (Maglischo, Maglischo and Santos, 1987). Using the centre of mass method would reveal some differences in the velocity-time curve, but its calculation is more complex and time consuming (Leblanc et al., 2007). There are still limitations in determining intra-cyclic velocity-time curves with the 3-D methodology as it depends on the accuracy of the anatomical model used to compute inter-limb inertial effects (Schnitzler et al., 2010; Winter, 2005). However determination of intra-cyclic velocity-time curves using a fixed point has been shown to be reliable (Costill, Lee and D'Acquisto, 1987).

There are inter-individual differences in linear velocity curves due to inter-individual differences in swimming technique (Barbosa et al., 2010). The breaststroke linear velocity curve is characterised by a bi-modal and uni-modal profile (Seifert, et al., 2010) (Figure 1.4).

**Figure 1.4** A comparison of breaststroke stroke cycle patterns and leg and arm phases one has two peaks of velocity (left panel) and the other has one peak of velocity (Seifert et al., 2010).

The first velocity increase is related to the leg action and the second velocity increase is related to the arm action (Bober & Czabanski, 1975; Kent & Atha, 1975; Figure 1.4). In the literature there is some ambiguity as to where peak velocity occurs during the stroke cycle. Some authors have reported peak velocity during the leg kick (Capitao et al., 2006; Craig, Termin and Pendergast, 2006; D'Acquisto and Costill, 1988; Yoshimurs et al., 2005), other authors have reported that the peak linear velocity occurs during the arm stroke (Maglischo, Maglischo and Santos, 1987; Manley and Atha, 1992), whilst others have reported similar peak values during the arm and leg phases of the stroke cycle (Bober and Czabanski, 1975; Kent and Atha, 1975; Tourny et al., 1992). The difference in the literature could be due to inter-individual differences in the groups of swimmers assessed as all individuals will present

different technical characteristics (Maglischo, 2003; Seifert et al., 2010). Breaststroke swimmers don't only present with different stroke co-ordination patterns but also can be characterised as arm or leg dominant swimmers (Maglischo, 2003; Seifert et al., 2010), thus altering the location of peak velocity within the stroke cycle.

Intra-cyclic linear velocity curves are generated from the analysis of changes in velocity. Changes in velocity occur when the propulsive force is greater than the resistive or drag force (Vilas-Boas, Fernandes and Barbosa, 2010). If propulsive force is greater than the resistive or drag force then the movement will be shown as acceleration. If the resistive or drag force is greater than the propulsive force then movement will be shown as deceleration. If the propulsive force is equal to the resistive and or drag force then the acceleration will be zero (Vilas-Boas 2011).

Drag force, which is primarily due to hydrodynamic drag during swimming, has been defined as an external force that acts on the swimmers body, parallel but, in the opposite direction to their movement direction (Figure 1.5) (Barbosa et al., 2011). Frictional drag or surface drag is developed when the water passes over a surface and is thus part of passive drag. Frictional drag is affected by the skin's roughness, body contouring, hair and swim suit material (Rushall et al., 1994; Toussaint et al., 2006; Zaida et al., 2008). Form drag is created by the shape of the swimmer and is also a component of passive drag. The largest factor affecting form drag is the cross-sectional area (projected surface area) that is presented to the oncoming fluid. Form drag is more important during high speed swimming as it increases by the square of the relative velocity (Rushall et al., 1994). Form drag becomes active drag when changes in the swimmers position are disadvantageous such a time is during the recovery of the legs within breaststroke stroke cycle. Wave drag is a form of active drag and occurs as a

result of the waves, wakes and turbulence that a swimmer or other forces generate (Rushall et al., 1994). Wave drag is a result of large vertical and lateral movements and represents the largest part of total drag (Marinho et al., 2009; Toussaint et al., 2006). Active drag is affected by changes in body size and velocity and has been shown to be significantly higher in adults compared to children (Kjendlie and Stallman, 2008).

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**Figure 1.5** The hydrodynamic drag acting on a breaststroke swimmer adapted from Maglischo, (2003).

The resistive force is dependent on the anthropometric characteristics of the swimmer, on the water field and on swimming technique (Barbosa et al., 2010). The hydrodynamic resistance of forward motion in a fluid medium can be expressed by Newton's equation (Equation 1.6).

Equation 1.6 
$$D = \frac{1}{2} C_D \rho S v^2$$

Where  $C_D$  represents the drag coefficient,  $\rho$  represents the fluid density,  $S$  represents the projected surface area of the swimmer and  $v$  represents the swimmers velocity. The determination of passive drag in swimming has been investigated by towing a non-swimming subject through water (Karpovich, 1933). However, passive drag does not consider the drag

that a swimmer creates when they produce thrust to overcome drag, which is known as active drag (Barbosa et al., 2010).

Attempts have been made to try and quantify active drag during swimming. There are three main methods that have been proposed. The first method uses interpolation techniques (e.g. (Clarys and Jiskoot, 1975; Di Prampero et al., 1974) that involve indirect calculations based upon changes in oxygen consumption as additional loads are placed upon the swimmer. The second method is the system that measured active drag (MAD-system) which relies on a direct measurement of force applied to force transducers in the water (Hollander et al., 1986). The MAD-system is limited to the analysis of the front crawl arm only technique. The third method is the perturbation method (Kolmogorov and Duplischeva, 1992). The perturbation method uses the difference in mean velocity between the average of two maximal effort lengths of swimming and two maximal effort lengths towing a hydrodynamic that has a known passive drag value, so that the added drag to the swimmer is known. The drag force is calculated from the difference in swimming velocity (Kolmogorov and Duplischeva, 1992). The perturbation method assumes that the power output used to overcome drag is maximal and constant. The perturbation method does allow the evaluation of passive drag in all four of the competitive strokes. There is also the active drag evaluation system (ADES) (Bideau et al., 2003; Nicolas et al., 2007) which is based on the velocity perturbation method (Kolmogorov and Duplischeva, 1992). The ADES system requires the swimmer to be attached via a non-elastic wire to an external device that controls the load. The ADES reduces the drag inaccuracies that are present in the velocity perturbation method (Bideau et al., 2003)

The acceleration ( $a$ ) of a swimmer is dependent on the change in velocity ( $v$ ) during a given period of time and is dependent on the force ( $F$ ) applied and the mass ( $m$ ) of the swimmer (Equation 1.7)

Equation 1.7       $F = ma$

The inertial term ( $m$ ) of the equation of movement is derived by adding the swimmers body mass ( $m_b$ ) to the added mass of water ( $m_a$ ) that is moved together (Vogal 1994). The resultant ( $F$ ) is the result of the vector adding of the propulsive force ( $P$ ) and the resistive force ( $D$ ) (Equation 1.8

Equation 1.8       $P + D = (m_b + m_a) .a$

The intra-cyclic linear velocity variations are a result of the prevalence of propulsive force or resistive force or are a consequence of an increased (or reduced) added mass effect during the stroke cycle. The assessment of the added mass during swimming is still an area that has not been well quantified and warrants further research (Barbosa et al., 2010).

Propulsion and drag have been shown, via the assessment of impulses generated during the different phases of the stroke, to change disproportionately during the breaststroke stroke cycle (Vilas-Boas, 1996). The assessment of impulses has been done using the equation (Equation 1.9) proposed by Vilas Boas, (1996). Where IVF = overall index of velocity fluctuation, Leg V = peak velocity during leg propulsion, ALL V = minimum velocity during the recovery phase, Arm V = peak velocity during arm propulsion, Arm Rec V = the minimum velocity during the arm recovery phase.

Equation 1.9

$$IVF = \frac{\text{Mean V}}{(\text{Leg V} - \text{ALL V}) + (\text{Arm V} - \text{Arm Rec V})}$$

The ability to generate high peak propulsion, mean propulsion and propulsive power values is considered a main characteristic with regards success in the short swimming events (Vilas-Boas, Fernandes and Barbosa, 2010) such that short distance swimmers are expected to produce high acceleration even when experiencing high drag forces. As drag is dependent on the square of velocity it is important to minimise intra-cyclic velocity fluctuations to minimise drag and maximise speed. However to date no research has been done that has investigated the changes in inter-velocity fluctuations with in a swim.

#### *1.4.2 Methods Used to Assess Intra Cyclic Variations in Swimming Velocity*

There are two methods used in the assessment of intra cyclic velocity; 1) the fixed method technique which is characterised by the evaluation of a fixed land mark, which is normally the hip joint; 2) the evaluation of the swimmer's overall centre of mass (CM)

The techniques used for the fixed point method are based on the use of kinematics of a fixed point and can be subdivided into image based and mechanical methods (Vilas-Boas, Fernandes and Barbosa, 2010). The mechanical methods include the use of cable speed meters. This work was started by Karpovich & Karpovich, (1970) who used a magnetic tape natograph. The use of the mechanical assessment has been redeveloped since the 1970's and is now used by a number of researchers in the assessment of intra-cyclic velocity (Leblanc et

al., 2007; Lima et al., 2006). The mechanical cable is normally attached to the hip region through the use of a belt attached to the swimmer (Lima et al., 2006) (Figure 1.6). The belt has not been reported to affect the performance of the swimmer (Leblanc et al., 2007). However, the limitation of this method is that it only allows the assessment of intra-cyclic velocity over a single length of a pool, as the swimmer must swim away from the velocity meter and constant tension must be maintain on the cable. The second main mechanical method is the propeller based speed meter, which was proposed by Kent & Atha, (1975) and has been further developed by others (Hahn and Krug, 1992; Manley and Atha, 1992). Other mechanical methods include usage of accelerometers (Holmer, 1979; Tella et al., 2008) and use of the Speed Capsule that has built in semiconductor tension-transforms that measure water pressure, which is suggested to be proportional to the swimmers' translational velocity (Boicev and Tzvetko, 1975).



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**Figure 1.6** The cable speed meter developed by Lima, et al., (2006) and an example of velocity-time output data, showing both the instantaneous velocity (black trace), and the mean velocity (blue trace) of the trial. The stick figure allows identification of the different phases of the breaststroke cycle.

Image based methods, used in the fixed point technique mainly involve the use of film or video that has to be digitised. The digitising process requires the digitisation of a fixed point, which is normally the hip joint. The time (t) changes in the hip joint can be determined and thus the velocity (V) can be calculated as distance (d) is known (Equation 1.10)

Equation 1.10

$$V = t/d$$

The limitations of the image based method include image distortions, water bubbles in front of the camera, waves distorting the image, especially where the two fluid mediums interact as the boundary layer of air and water (Vilas-Boas, Fernandes and Barbosa, 2010). Problems are also caused by parallax, digitising and calibration errors. One of the issues with the digitising method is the time it takes to process the data. This is another drawback with regards to this system as the ability to provide real time feedback is lacking as automated tracking is not currently available for dual media images (above and below water). However, 2D video analysis does have the advantage, over the mechanical devices, of allowing the user to track the swimmer throughout a complete swim. Therefore this technique allows the investigation of inter-lap and intra-lap changes in stroke kinematics along with changes in propulsive and resistive force, which thus far has not been investigated in breaststroke swimming.

The assessment of the centre of mass (CM) is conducted via analysis of 3D movements of the swimmer and is reported (Figueiredo et al., 2009; Psycharakis and Sanders, 2009) to be a more accurate method of assessing intra-cyclic velocity of a swimmer than the fixed point methods previously discussed. The limitations of the 3D assessment is the time it takes to analyse the data, and reliance on the accuracy of the anthropometric biomechanical methods used (Schnitzler et al., 2010; Winter, 2005). The centre of mass assessment is also affected by the same issues as 2D video analysis regards filming in the two mediums of air and water, which can cause issues with image quality, parallax errors, digitising and calibration issues and it uses a fixed section of the pool so does not allow full tracking of a whole swim. The 3D reconstruction method of CM is reported (Figueiredo et al., 2009; Psycharakis and Sanders, 2009) to be a more valid method for the assessment of swimming kinematics. It has been shown that there are differences in the displacement of the hip and

CM as the hip does not account for the relative movements of other parts of the body (Barbosa et al., 2003; Barbosa et al., 2003; Maglischo, Maglischo and Santos, 1987). Previous studies have shown that the velocity time curves of the hip joint in breaststroke swimming are shifted by 3-5 % earlier than for CM. There have been higher reported velocities from the analysis of the hip compared to that of the CM (Leblanc et al., 2007). However the coefficients of correlation between the displacement curves of the two methods have been shown to be significant as the curves follow similar patterns (Maglischo, Maglischo and Santos, 1987).

## **1.5 Neuromuscular Activity during Swimming**

The first studies regarding neuromuscular activity in swimming quantified muscle activity in the early sixties (Ikai, Ishii and Miyashita, 1964). Ikai, et al., (1964) reported that the bicep brachialis, triceps brachialis and deltoid were highly active during the strokes. Ikai, et al., (1964) reported that the elbow flexors presented lower levels of activity compared to the elbow extensors in the breaststroke, front crawl and butterfly strokes.

In the late sixties the trend in electromyography (EMG) studies was to focus more on quantifying the EMG signal. This trend was started by Lewillie, (1973) and followed by Clarys, (1983). Compared to kinematic and kinetic analyses neuromuscular assessments are less well represented in swimming research literature. The low number of swimming EMG studies is in part due to the aquatic environment which makes the collection of EMG signals even more complicated than it normally is (Clarys, 1988; Clarys and Cabri, 1993). One of the main challenges is preventing the water from interfering with the recording of the electrical signal from the muscle (Masumoto and Mercer, 2008).

### *1.5.1 Generation of the EMG Signal*

The measurement of neuromuscular activity requires the measurement of the electrical signal that is generated to induce muscular contraction (Basmajian and De Luca, 1985). The excitation of muscle requires an electrical signal to be transmitted along an axon (Figure 1.7) (stimulated by the central nervous system or a reflex) of the involved neurons, to the motor end plate (Figure 1.7) where an end plate potential is formed where the fibre innervates with the motor neuron known as the neuromuscular junction. This causes a membrane depolarisation which is immediately restored by a repolarisation phase which is followed by an after hyperpolarisation period of the membrane (Figure 1.8). The process involves the movement of ions across the axolemma and sarcolemma membranes, which creates an electrical signal (action potential) that travels along the muscle membranes leading to the release of calcium within the muscle fibre (Enoka, 2002) which can be detected by the surface electrodes.

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**Figure 1.7** The motor neuron and neuromuscular junction (NMJ). The NMJ marks the end of the axon and forms a synapse with skeletal muscle. Synaptic vesicles migrate to the terminal membrane and release neurotransmitters into the synaptic cleft via exocytosis where they bind to receptors on the surface of the sarcolemma membrane (Bear, Connors and Paradiso, 2001)

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**Figure 1.8** Schematic diagram of the action potential (Enoka, 2002).

#### *1.5.2 Method for Detecting the EMG Signal*

As a result of the electrical conductivity of the tissue surrounding muscle an electrical signal can be detected; this is known as the EMG signal (Clarys & Rouard 2010). The EMG signal can be recorded on an electrode either by using fine wire or needle electrodes implanted into the muscle, or by using an electrode secured to the surface of the skin, better known as surface electromyography (sEMG).

Needle and fine wire electrodes are inserted into the muscle of interest (Kamen and Gabriel, 2010). Needle electrodes are used for the analysis of motor unit action potentials (MUAP); they also allow the analysis of firing rates and recruitment of muscle fibres. Therefore, needle electrodes are used in the analysis of control properties. The fine wire electrodes are used for the analysis of deep muscle, both in kinesiological and neurophysiological studies (Kamen and Gabriel, 2010).

There are two types of electrode used in surface electromyography (sEMG), these are passive electrodes and active electrodes (Clarys et al., 2010; Kamen and Gabriel, 2010). Passive electrodes usually consist of a metal surface, that is the conductive detection surface, that senses the current on the skin through the skin electrode interface (Kamen and Gabriel, 2010). Active electrodes contain a high input impedance electronic amplifier in the same housing as the detection surface (Kamen and Gabriel, 2010). The advantage of the active electrode over the passive electrode is the decline of erroneous registrations (Clarys et al., 2010). There have been reported concerns of reduced detectable electrical output whilst recording muscle activity of the human muscle in the aquatic environment (Clarys, 1985). However, preceding studies have reported (Masumoto and Mercer, 2008; Rainoldi et al., 2004; Veneziano et al., 2006) that with effective waterproofing there is no difference in the electrical output of the human muscle. The disadvantages of surface electrodes are that they can only be used effectively with superficial muscles and cannot be used to selectively detect signals from small muscles. The active electrode has been reported (Clarys et al., 2010; Masumoto and Mercer, 2008) to be the most reliable electrode for the use of analysis of complex dynamic sport and exercise movements.

### *1.5.3 Methodological Consideration of EMG Analysis in the Aquatic Environment*

Several methodologies have been proposed to allow the recording of EMG signals in the aquatic environment (Caty et al., 2007; Masumoto et al., 2004; Masumoto et al., 2005; Mercer et al., 2005). These methods can be categorised into two general approaches: 1) to create a localised waterproof seal around the leads (Caty et al., 2007; Masumoto et al., 2004; Masumoto and Mercer, 2008), and 2) to create a whole-body waterproof system via the subjects wearing a dry suit (Mercer et al., 2005).

To create a localised waterproof environment around the electrodes and the EMG leads it is common to apply an adhesive waterproof tape over the electrodes (Figure 1.9 a, b). Some authors (Masumoto and Mercer, 2008) have also recommended the use of a foam pad outer covering (Figure 1.9 c). The taping should be done in a manner that does not interfere with the normal movement of the studied muscles (Masumoto & Mercer 2008). It has been recommended (Masumoto & Mercer 2008) that when using a localised waterproof environment that the EMG signal should be monitored for changes that indicate water leakage, such as a band of high-frequency noise throughout the recorded signal. One of the advantages of using this methodology is that it only requires water proofing of localised areas.

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**Figure 1.9** Waterproof dressing over the surface electromyography (sEMG) electrodes: placed electrodes (A), covered with adhesive tape (B), and covered with foam pads (C). (Masumoto and Mercer, 2008).

To create a whole-body water proof system subjects are required to wear a dry suit (Mercer et al., 2005). The dry suit consists of a waterproof material, with a tight seal around the arms, legs and neck. The EMG leads are protected from the water as the body is kept completely dry. A limitation of this system is that it is unknown how the dry suit influences



swimming movements. Another limitation of this system is the need for investigators to have a large array of dry suits to fit different sizes of subjects (Masumoto and Mercer, 2008).

Another concern in measuring EMG in the aquatic environment is the use of electronics when immersed in water (Masumoto and Mercer, 2008). There are two main types of EMG systems available, the tethered and the telemetry. It has been reported (Clarys, et al., 2004) that there are no differences between the two methods so it was concluded that muscle activity could be recorded during immersion with a telemetry system (Figure 1.10). Using telemetry EMG systems reduces the risk of injury to the participants, as units are powered via battery (Masumoto and Mercer, 2008).

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**Figure 1.10** Water decreases the amplitude of conventional online and telemetric EMG signals when compared with signals recorded in air (Clarys, 1988; Clarys and Rouard, 2010).

There are a number of concerns with the reliability of the EMG signal that is detected using sEMG: signal to noise ratio (De Luca, 1997); positioning of the electrode (Clarys and Cabri, 1993). The signal to noise ratio is the ratio of the energy in the EMG signal to the energy in the noise signal. Noise in the EMG signal can originate from various sources, such as: inherent noise in the electronic components in the detection and recording equipment; reformat paragraph to put rest of list here, then discuss each in turn. Noise in the EMG signal cannot be eliminated, but it can be reduced by using high quality electronic components. The noise that is present in the EMG signal can originate from; ambient noise which is due to electromagnetic radion; motion artefacts, this noise comes from either the interface between the detection surface of the electrode and the skin. This noise can be reduced through correct preparation of the skin. It has been recommended that the skin is shaved using a dry razor to remove the dead dermis and that the skin is subsequently rubbed with an alcoholic solution (SENIMA; Hermens & Freriks, 1997). Noise due to motion artefacts is related to movement of cables that connect to the electrode. The amplitude of the EMG signal is quasi-random in nature meaning that the EMG signal is unstable due to the qusi-random nature of the firing rate of the motor units. Other factors that need to be considered with sEMG are the position of the surface electrode on the muscle. The electrode should be placed in the middle of the muscle belly along its midline and should be aligned parallel to the length of the muscle (SENIMA; Hermens & Freriks, 1997). The electrode should not be placed on or near the tendinous insertion as this will reduce the amplitude of the EMG signal (Clarys and Cabri, 1993).

#### *1.5.4 Signal Processing*

The EMG signal is an expression of the dynamic involvement of particular muscles within a determined range. EMG signals can either be treated according to amplitude or frequency to obtain quantitative information about active muscles (Clarys and Rouard, 2010). The amplitude process is initiated with the full-wave rectification of the raw EMG signal which returns the absolute values of the EMG signal (Figure 1.10). This is followed by an averaging graphical linear representation known as the linear envelope (Clarys and Rouard, 2010). All EMG signals need to be filtered and verified against artefacts such as noise, baseline deviations, heart-beat etc (Figure 1.11).

The integral of the EMG signal can be calculated in a number of different ways but in the study of sports and occupational movements the surface under the linear envelope curve is used (Clarys and Rouard, 2010). The integrated EMG (iEMG) is the expression of the muscular intensity which is related to the number of recruited motor units and their synchronisation (Clarys and Rouard, 2010).

Different factors can alter the amplitude of the EMG signal such as the size of the muscle, the type of muscle fibres, the nature of the interface between the skin and the surface electrodes, the placement of the electrodes and the amount of adipose tissue under the skin (Hermens and Freriks, 1997; Solomonow et al., 1988). In addition to this each and every individual will present different basic electrical tonus and for all these reasons the EMG signal has to be normalised (Clarys and Rouard, 2010).

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**Figure 1.11** Basic signal processing of EMG within swimming and sports in general (Clarys and Rouard, 2010).

#### *1.5.5 Normalisation*

Variability occurs in the EMG signal as no two EMG profiles will be identical due to changes in the motor units controlling the action (Ball and Scurr, 2010; Burden, 2010; Clarys et al., 2010). As a consequence of this variability in the EMG signal normalisation techniques have been developed to allow comparison between subjects and between trials (Ball and Scurr, 2010; Burden, 2010; Clarys et al., 2010). The common normalising factors are either the EMG of the maximal effort or the highest EMG (Ball and Scurr, 2010; Burden, 2010; Clarys et al., 2010). The subject is required to complete a maximal voluntary contraction (MVC) of the muscle being studied. Usually the MVC is analysed from a static (isometric) contraction (MVIC), however, it can also be measured from a dynamic muscle action in accordance with SENIAM (Merletti et al., 1999). The MVC amplitude, which can either be raw or rectified (Clarys et al., 2010), is then used as the reference value (100%). This method of normalising the EMG signal is very popular and is acceptable in static contractions (Clarys

et al., 2010). However, the use of the isometric maximal voluntary contraction (MVIC) as a point of reference for dynamic activities is questionable (Clarys, 2000; Clarys et al., 2010). Different maximal EMG values will be found at different joint angles of movement, and different maximal EMG values will be found within the same subject repeating the same task on different occasions. It may also be difficult to get the subject to complete an MVIC correctly, due to level of experience. There is also the question of linearity when values during dynamic sporting movements exceed 100 % of MVC such as Clarys, et al., (1983) who reported dynamic percentages of 160 % in swimming, while Jobe, et al., (1984) reported up to 226 % of MVC in baseball pitching. However, the MVIC method is still used to normalise EMG signals during dynamic actions as there is still no consensus on the appropriate methods for normalisation (Chapman et al., 2010; Nishijima et al., 2010; Rouffet and Hautier, 2008).

Alternative methods for normalisation have been developed for use in sport, exercise and occupational kinesiological studies (Ball and Scurr, 2010; Burden, 2010; Clarys et al., 2010). These alternative methods involve: i) normalisation to the highest peak activity ( $Peak_{Task}$ ) activity in dynamic conditions; or ii) normalisation to the mean EMG ( $Mean_{Task}$ ). Both of these normalisation values are usually obtained from an ensemble average rather than a single trial EMG found within each test/exercise trial (Burden, 2010; Clarys et al., 2010). Research has suggested that the use of either  $Peak_{Task}$ , or preferably  $Mean_{Task}$  can be used if the aim of the normalisation is to maximise the reduction of EMG variability between subjects (Burden and Bartlett, 1999; Knutson et al., 1994; Yang and Winter, 1984). These methods of normalisation remove the true biological variation in the signal and improve its homogeneity (Allison, Marshall and Singer, 1993; Knutson et al., 1994). However, this is also true to a lesser extent in all methods of normalisation (Burden, 2010).

### 1.5.6 EMG Signal Time Domain Processing

EMG signals have been processed in a number of ways since the introductions of computers. Nowadays there are a number of ways to analyse and quantify muscle activity or investigate how activity changes over time. Raw EMGs are processed in what is known as the time domain. This can be done either by using the average rectified EMG (ARV) (Equation 1.11)

Equation 1.11 Average rectified EMG (ARV)

$$ARV = \frac{1}{T} \sum_{t=1}^T |EMG(t_i)|.$$

Where  $|EMG(t_i)|$  is the absolute value of EMG signal in the data window and  $T$  is the time over which ARV is calculated

The calculation of the ARV EMG involves the reversing of all the negative phases of the raw EMG (full wave rectification). The integral is then calculated over a time period or window ( $T$ ) and the resulting integrated EMG (iEMG) is divided by the time window from the ARV (Equation 1.11). The root mean square (RMS) is the square root of the average power of the raw EMG calculated over a specific period or window ( $T$ ) (Equation 1.12)

Equation 1.12 Root mean squared (RMS)

$$RMS = \sqrt{\frac{1}{T} \sum_{t=1}^T EMG^2(t_i)}.$$

Where  $EMG^2(t_i)$  is the squared value of each datum of EMG in the data window

Both methods are recognised as acceptable methods and are commonly used in EMG signal analysis (De Luca and Van Dyk, 1975; Farina and Merletti, 2000). As well as a single calculation of the RMS or the ARV the raw EMG signal can be processed by using a moving average. The moving average time period can vary from 10 to 200 ms depending on the type of investigation taking place. A time period of 10-50 ms is used for rapid alterations in EMG. A longer time window 100-200 ms will reduce the variability of the peak amplitudes but will lose the trend of the new EMG. A possible solution is to utilise an overlapping time window. Although these are recognised methods there are limitations with these methods with respect to the analysis of the EMG amplitude. Using single maximal values for RMS is not a robust method against movement artefacts. The averaging analysis over a selected time window provides a more robust measure against movement artefacts, however it is less sensitive to changes in the EMG signal and may mask differences in muscle activation intensities between experimental conditions.

The linear envelope can also be used on dynamic contractions as it is thought to provide specific information regarding how a muscle is controlled (Kamen and Gabriel, 2010). The linear envelope is a similar method to the moving average, however, linear envelope involves the smoothing of the rectified EMG with a low pass filter such as a Butterworth filter with a cut-off between 3 and 80 Hz. The cut off is similar to the time window width. A lower cut-off frequency will result in a smoother curve whereas a higher cut-off frequency will allow the detection of more rapid changes in the EMG signal.

The processed EMG signal can be used to estimate when a muscle is on (active) or off (inactive) (Hodges and Bui, 1996). This process is better known as the determination of EMG onset and off set (Hodges and Bui, 1996). The EMG onset can be determined by manual

detection which remains the gold standard (Hodges and Bui, 1996). The criteria for the onset is normally composed of two requirements: (i) that a baseline is set, above which EMG activity is detected. This baseline has previously been set at 10 % of the maximum value for that particular muscle activity (Baum and Li, 2003). However this threshold is not fixed and can be moved upwards to 20 % (Baum and Li, 2003); (ii) that EMG activity must exceed the baseline for a critical time period (Hodges and Bui, 1996). This second criteria minimises the false detection of erratic departures from the baseline which have been show to occur prior to muscle contraction (Walter, 1984); Figure 1.12)

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**Figure 1.12** The onset of the linear envelope – detected EMG burst of muscle activity. The first criterion for the determination of onset is the threshold amplitude ( $A_c$ ) (dotted line) that the muscle activity must exceed. The second criterion is the critical time period ( $t_c$ ) during which the EMG burst must stay above the threshold amplitude. The smaller burst does not meet both criterion. (Walter, 1984)



### 1.5.7 EMG Signal Frequency Domain Processing

Frequency domain analysis presents the data as a function of frequencies. The most commonly frequency domain analysis used in biomechanics is the Fourier transform (Giakas, 2004). The Fourier transform is a mathematical procedure used to describe complicated analog signals with the use of sine and cosine functions. First the signal needs to be converted from the analog signal into a digital signal and this is done using a discrete Fourier transform (DFT). The DFT (Cooley and Turkey, 1965) calculates the contribution of the sinusoidal components to reconstruct the signal. The DFT is calculated from the harmonics which constitute the signal. The individual harmonics ( $\alpha_k$ ) of a signal can be calculated using equation 1.13: where  $W = e^{2j\pi/N}$ ,  $[n]$  represents the  $n^{\text{th}}$  data point,  $\pi = 3.1415\dots$ ,  $N$  is the number of data,  $k$  is the harmonic number, and  $n$  is the data index point. Equation 1.13 allows the transformation of the time domain to the frequency domain through the calculation of the Fourier coefficients ( $\alpha_k$ ) (Giakas, 2004). This is a relatively slow process for calculating Fourier coefficients (Brigham, 1974)

$$\text{Equation (1.13)} \quad \alpha_k = \sum_{n=0}^{N-1} x[n] W^{kn}$$

Faster approximations have been developed that use fewer calculations these algorithms are referred to as Fast Fourier Transforms (FFT). This process is faster because the segment data used for the analysis is limited to the power of 2 (e.g. 256, 512, 1024 or 2048 points) (Kamen & Gabriel, 2010). If all the individual harmonics ( $\alpha_k$ ) are known then it is possible to synthesise the signal precisely, thus transforming the signal from the time to the frequency domain (Giakas, 2004) with equation 1.14; where  $W = e^{2j\pi/N}$ ,  $\alpha_k$  represent the  $k^{\text{th}}$

harmonic,  $\pi = 3.1415.....$ ,  $N$  is the number of data,  $k$  is the harmonic number, and  $n$  is the data index point. This process is known as the inverse DFT (Giakas, 2004).

$$\text{Equation (1.14)} \quad x[n] = \frac{1}{N} \sum_{k=0}^{N-1} a_k[n] W^{kn}$$

The output of the FFT is typically represented as the power spectrum density (PSD) which shows the relative magnitudes of the range of frequencies represented in the raw signal. The PSD represents the square magnitude of the signal's Fourier Transform

One of the two parameters that are commonly obtained from the PSD, in order to quantify it are: the median frequency (MDF) (Equation 1.15), which is defined as the frequency that divides the PSD into equal halves; the mean power frequency (MPF) (Equation 1.16), which is calculated as the sum of the product of the individual and their power divided by the total power.

Equation 1.15

$$MDF = \frac{1}{TP} \sum_{k=0}^{N-1} (f[k] \cdot PSD[k])$$

Where  $k$  is the index for the frequencies  $\omega[k] = 2\pi k / N$ :  $k=0, 1, ..., N-1$ .

Equation 1.16

$$0.5 = \frac{1}{TP} \sum_{k=0}^{MDF} PSD[k]$$

Whilst the MPF is less variable than the MDF the latter is less sensitive to noise and more sensitive to spectral compression (Merletti et al., 1999). It is recommended SENIAM (Merletti et al., 1999) that the MDF can be used as an indicator of muscle fatigue. It is recommended that MPF and MDF are reported together as they provide overlapping and different information (Farina and Merletti, 2000). Changes in MPF and MDF are both highly correlated, consequently only one of these needs to be reported to describe fatigue changes. In the swimming literature all the studies that have reported fatigue changes (Caty et al., 2007 and Strin et al., 2011) have reported the findings as changes in MPF. When interpreting finding of different studies it is a limiting factor that different methods have been utilised in the analysis of muscle activity. Thus it is important to use similar methods to those of studies in the same area.

Raw EMG's are processed in the frequency domain primarily to investigate changes in the signal that accompany fatigue. It is well established (Bonnard et al., 1994; Ikuta et al., 2012; Ng and Richardson, 1996; Potvin, 1997) that fatigue is associated with changes in the EMG signal shown as a compression of the frequency spectrum to lower frequencies as a result of a decrease in the conduction velocity (CV) of the action potentials (AP).

Regardless of the parameter used to analyse the PSD, it is typically obtained from consecutive time windows, to enable the analysis of the changes that occur in the signal in order to monitor the changes due to fatigue. Successive values from the contraction period are then analysed using linear regression; with the intercept of the regression line being the initial frequency and the gradient representing the fatigue rate (Ng and Richardson, 1996).

When investigating dynamic contractions although using the FFT is widely accepted as a method for processing the EMG signal (Frere et al., 2011) there is a problem with the stability of the signal, due to the recruitment and de-recruitment of motor units (MU). It is recommended if using FFT on dynamic movements (De Luca, 1997) that the calculation of MDF and MPF should only be done at the same phase of the repetitive cycle. The limitations of obtaining spectral parameters from non-stationary signals has largely been overcome by using the joint time frequency domain approach, which estimates the change in frequency as a function of time (Giakas, 2004). The joint time frequency domain approach can be achieved using a short-time Fourier transform which: splits the EMG into small continuous, or overlapping time windows; applies a FFT to each; and calculates MPF or MDF (Kamen and Gabriel, 2010). There are more sophisticated FFT available such as: the Wigner-Ville transform from which instantaneous MDF or MPF can be calculated (Knaflitz and Bonato, 1999); the Hilbert transform from which the average instantaneous frequency is obtained (Georgakis, Stergioulas and Giakas, 2003); and wavelet transform analysis which produces intensity spectra (Wakeling et al., 2001). There are limitations with wavelet transform analysis as there is a lack of consensus with regards the techniques used with respect to the mother wavelet and the scale functions used (Frere et al., 2011). The wavelet transform has been used to clarify the relationship between muscle fatigue, muscle strength and the EMG signal (Frere et al., 2011).

#### *1.5.8 EMG Studies in Swimming*

There are two main areas of research in swimming that have been addressed using EMG. These are qualitative and quantitative. The quantitative studies have used the EMG in the time domain to describe muscle activity. This allows the duration of the muscle activity to

be determined via detection of when a muscle is active or non-active. Lewillie, (1973) conducted a case study on the four strokes at three swimming intensities which were slow, medium, and fast. The highest neuromuscular activity was recorded in the butterfly stroke at the fast intensity. The increase in activity from slow to fast lead to increased activity in anterior deltoid and triceps surae across all strokes. Nuber, et al., (1986) also reported higher activity of the pectoral major and the latissimus dorsi and reported that they were the main muscles used during the pull phase of the front crawl, breaststroke and butterfly strokes. In breaststroke swimming there are only three studies that have report the activity of muscle of the upper and lower body (Tokuyama, Okamoto and Kumamoto, 1976; Yoshizawa et al., 1976; Yoshizawa et al., 1978). It was reported that the major leg muscles that are used in the breaststroke leg kick are the vastus lateralis, vastus medialis, rectus femoris and biceps femoris. The authors also reported the important muscles of the upper body that are used during breaststroke as the deltoid, pectoralis major, latissimus dorsi, biceps brachii and the triceps brachii muscle. This finding is supported by the kinesiological analysis (Table 1.2 and 1.3) that was carried out by Rodeo, (1984).

**Table 1.2** Kinesiological analysis of the muscles used during the catch, down-sweep, insweep and recovery phases of the arm stroke in breaststroke (Rodeo, 1984).

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**Table 1.3** Kinesiological analyses of the catch, down-sweep, insweep and recovery of the breaststroke leg kick (Rodeo, 1984).

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The quantitative approach to EMG uses the assessment of the EMG signal with regards spectral analysis. This allows, as discussed earlier, the analysis of change in frequency over time. Monteil, et al., (1996) analysed the change in muscle activity at the beginning and end of a 400 m front crawl swim in a flume. Monteil, et al., (1996) reported a decrease in activity during the insweep and an increase during the outstroke. The authors concluded that there was a shift in the force production from the insweep to the outstroke and as a subsequent decrease in hand velocity. Aujouannet, et al., (2006) reported a similar shift

to lower EMG frequencies in the biceps brachii and triceps brachii muscles before and after a 4 x 50 m swimming test. Caty, et al., (2007) reported a decrease in EMG frequency in two forearm muscles carpi ulnaris and flexor carpi ulnaris during a 4 x 50 m front crawl swim.

## **1.6 Fatigue**

Although it is quite easy to know when one is suffering from fatigue it is entirely another matter to be able to ascertain the physiological mechanisms that are responsible for the onset of fatigue. Even though the study of fatigue has been around since the work of Angelo Mosses in the late 1800's (Di Giulio, Daniele and Tipton, 2006) we are still unable to state for certain why individuals become fatigued under certain circumstances (Enoka and Duchateau, 2008). The term fatigue has been used to symbolise a temporary decrease in the capacity to perform physical actions (Allen and Westerblad, 2001; Friedman et al., 2007). Skeletal muscle fatigue as a result of high intensity exercise is extremely complex, multi-factorial and is still not fully understood (Noakes, 2000; Allen, Lamb and Westerblad, 2008). Furthermore, there are numerous possible contributory factors that vary and interact differently between individuals based on genetics, environmental conditions, training status / methods and dietary manipulations.

Fatigue can be used to describe a reduction in the execution of motor actions, which results in a reduction in force capacity of the working muscles. Fatigue can be measured as a decrease in force production and/or a change in the electromyographic activity of the muscle(s) (Enoka and Duchateau, 2008). One of the most commonly used definitions of fatigue is the exercise induced reduction in the ability to maintain force or power (Bigland-



Ritchie and Woods, 1984) . Fatigue can start as soon as the form of exercise begins, so there is a gradual change in the force and power producing capacity of the working muscles.

During sporting events motor co-ordination and motor skills are crucial for performance and success. Previous research (Forestier and Nougier, 1998) has investigated the effects of fatigue on motor co-ordination in elite athletes; the authors reported that as levels of fatigue increased there were subsequent modifications in motor co-ordination, which enabled the athletes to maintain high performance levels. Similar findings were reported by Aune, et al., (2008) who proposed that elite athletes are able to adapt to the effects of fatigue more proficiently than beginners. Elite athletes are more effective at modifying co-ordination to meet the demands of fatigue, thereby preventing large decreases in overall performance. This has been shown in studies on the rowing ergometer (Turpin et al., 2011; Lay et al., 2002), cycling (Wakeling, Blake and Chan, 2010) and running (Slawinski et al., 2008).

The study of the affects of fatigue on swimming performance is limited with no studies, to the author's knowledge, in breaststroke swimming. A decrease in swimming velocity has been widely reported in swimming races (Caty et al., 2006; Craig et al., 1985) and in experimental trials (Craig et al., 1985; Tella et al., 2008; Weiss et al., 1988). The decrease in swimming velocity has been attributed to modifications in stroke parameters. Most of the studies have reported a decrease in stroke rate (Berger, de Groot and Hollander, 1995; Caty et al., 2006; Tella et al., 2008). This decrease in stroke rate has been reported (Keskinen and Komi, 1993) to be related to a reduction in the neural activation. This has been validated to a degree by Caty et al., (2007) who detected a decrease in the EMG frequency of the forearm muscles during a 4\*50 m maximal swim. A decrease in swimming velocity has also been attributed to alterations in the hands path (Alberty et al., 2003; Tourny-Chollet,

Seifert and Chollet, 2009). The authors described an increase in the duration of the catch, outstroke and insweep of the participants. This has also been shown in lower level front crawl swimmers (Alberty et al., 2003; Tourny-Chollet, Seifert and Chollet, 2009) with an increase in the time spent in the propulsive phases of the stroke. Hand velocity has been shown to correlate with decreases in swimming velocity (Monteil et al., 1996).

A decrease in force production has been reported in dry land, full tethered or half tethered front crawl swimmers (Rouard et al., 2003) in the swimming flume (Monteil et al., 1996) and on the MAD system measurement (Toussaint et al., 2006). When fatigued the front crawl swimmers were shown to have a reduced power output capacity (Rouard et al., 2003). This was reported (Rouard et al., 2003) following a maximal 100 m front crawl swim on the MAD system which was also associated with a decrease in swimming velocity and stroke rate (Toussaint et al., 2006). Large individual variation was observed in the fresh and fatigued conditions with respect to the glide phase (Alberty et al., 2003), insweep and upstroke phases (Alberty et al., 2003) of the front crawl. Understandings that there is individual variability with respect to adaptations to the phases of the stroke are valuable to evaluate kinematic and kinetics in swimming.

There are a limited number of studies that have investigated the effects of fatigue on EMG parameters in swimming with all the studies currently been carried out in front crawl swimming (Monteil et al., 1996; Rouard, Quezel and Billat, 1992; Aujouannet et al., 2006; Bigland-Ritchie and Woods, 1984; Wakayoshi et al., 1994; Strin et al., 2011; Ikuta et al., 2012). A study in front crawl swimming (Monteil et al., 1996) reported that when swimmers were fatigued there was a loss of specificity of the internal and external rotator muscles of the shoulder compared to the fresh condition. This was indicated by an increase in the IEMG in

the fatigued condition. This has been supported by the findings of Wakayoshi et al., (1994) who also reported increase in IEMG. These findings have not been supported by the findings of Rouard, et al., (1992) who reported no change in the IEMG in front crawl swimmers in 4\*100 m with 45s rest between swims suggesting that the test did not invoke exhaustion in the muscles. Consequently for a given exercise the effect of fatigue depends on the muscle involved. A decrease in the amplitude of the IEMG reflects a maximal effort, when there is an increase in the amplitude of the IEMG this indicates a submaximal effort and no change in amplitude equate to a low request of the muscle (Rourad, 2011).

Other types of EMG analysis have been used in swimming that has involved the analysis of the data with respect to the maximal voluntary contractions (MVC). It has been reported that (Aujouannet et al., 2006) there is a decrease in the MVC when assessed after an maximal effort front crawl swim of 4\*50 m with 10 seconds rest between trials. A decrease in the mean power frequency (MPF) for the muscles biceps and triceps brachii was reported thus suggesting a compression of the power spectrum to lower frequencies as a result of fatigue (Bigland-Ritchie and Woods, 1984). Utilising the same protocol Caty, et al., (2007) evaluated the time-frequency contents of the muscles flexor and extensor carpi ulnaris. The muscle activity was analysed using a time frequency algorithm that produced the instantaneous mean frequency (MNF) of the power spectrum density (Molinari et al., 2006). The findings of this study showed a significant decrease in the MNF for both the muscles at the 50 m swim, with a significant decrease in the IEMG for flexor carpi ulnaris thus indicating fatigue. The fatigue of these muscles were associated with a decrease in the wrist fixity. A study by Ikuta, et al., (2012) again utilised the same protocol as Caty, et al., (2007) in 200m front crawl swimmers however they recorded the sEMG of eleven muscles of which seven were of the upper body and four of the lower body. Only mean amplitude was reported

in this study which is a limitation, as need to consider both frequency and time spectrum if going to analyse fatigue. The results of this investigation showed a significant decrease in mean amplitude value (MAV) of the flexor carpi ulnaris, biceps and triceps brachii along with a decrease in the rectus femoris muscle and a decrease in swimming velocity. In contrast the rate of change in the MAV of the pectoralis major and latissimus dorsi muscles significantly correlated with the rate of change in swim velocity. Ikuta et al., (2012) reported that this decrease in swim velocity was related to a decrease in the activities of several muscles that co-ordinate with each other and that compensatory mechanism occurred between the pectoralis major and the other muscles in the final 50 m. The only study, thus far, that has investigated the change in muscle activity during a swim was Strin, et al., (2011) who reported a significant decrease in MPF of the muscles latissimus dorsi and triceps brachii of 20 -25 % during a maximal 100 m front crawl swim.

A fatigued swimmer, who is unable to maintain high levels of force production will experience a decrease in stroke length, an increase in stroke rate and an overall decrease in clean swim speed in front crawl swimmers (Strin et al., 2011). A decrease in the force, and or power, producing capacity during maximal explosive activities may be due to changes in inter-muscle co-ordination (Billaut, Basset and Falgairette, 2005), which may result in changes in the muscle activity of the major muscles used in breaststroke (Table 1.2 and 1.3) and therefore could result to changes in the co-ordination of the arms and legs. Muscle co-ordination has been defined as a distribution of muscle activation or force among individual muscles to produce a given combination of joint movements (Prilutsky, 2000). Muscle co-ordination has been investigated in rowing (Turpin et al., 2011) who reported increase in power was accompanied with an increase in muscle activity, similar changes in muscle

activity were reported in cycling (Dorel et al., 2012) with changes in the relative contributions of the muscles as power output changed.

A greater understanding of the changes that occur within a swim with regards to co-ordination of the arm and leg phases, the changes in the force and power production and the changes in the muscle co-ordination and activation levels would provide sports scientists, coaches and swimmers with greater insight into swimming performance. This information could assist with designing interventions to improve overall swimming performance and improving understanding of the risk of injury within the sport of swimming.

The muscular fatigability is specific to the muscles, the exercise and the subjects. Through the use of EMG studies we are able to evaluate individual strategies of swimming and the adjustments caused by fatigue (Rouard, 2011). There is still a need for further research into load sharing across muscles and to determine the central and peripheral components of fatigue in swimming. The quantification of the evolution of muscle fatigue could be a valuable tool in the improvement of specific training protocols (Rouard, 2011).

In summary, the main aims of the thesis were to understand the biomechanical and physiological changes that occur during a 100 m breaststroke swim. This will be achieved by meeting the following specific objectives:

#### Objectives

- 1) To evaluate stroke kinematics stroke rate, stroke length and linear velocity and arm-leg co-ordination over the duration of a 100 m breaststroke swim in

2) To establish sex differences in stroke kinematics over the duration of a 100 m breaststroke swim.

3) To evaluate intra-cyclic velocity fluctuations measured from a single point over the duration of a 100 m breaststroke swim;

4) To evaluate amplitude and frequency of the surface electromyography of the major muscles of the lower, trunk and upper body muscles during a 100 m breaststroke swim.

## **CHAPTER 2**

### **CHANGES IN KINEMATICS AND ARM-LEG CO-ORDINATION DURING A 100 M SHORT COURSE BREASTSTROKE SWIM**

## CHAPTER 2

### 2.0 Changes in Kinematics and Arm-leg Co-ordination during a 100 m Short Course Breaststroke Swim

#### 2.1 Abstract

This study investigated changes in kinematics, including arm-leg co-ordination, over four laps of 100 m short course breaststroke swim from a water start in twenty six (eight females and eighteen males) specialist breaststroke swimmers, FINA points  $567 \pm 110$ . Arm-leg co-ordination was defined using two co-ordination phases that described the continuity between the recovery and propulsive phases of the arms and the legs: CPhase1, corresponding to the time between the end of the leg kick and the start of the arm pull phases; and CPhase 2, corresponding to the time between the end of the arm pull phase and the start of leg kick phase. The duration of the four stroke phases, (Arm pull, arm recovery, leg kick and leg recovery), the duration of the two co-ordination phases, clean swim speed (CSS), stroke length (SL), stroke rate (SR) and stroke index (SI) were analysed during the last three strokes of each lap that were not affected by turning or finishing techniques. Each lap was recorded using three 50 Hz underwater cameras. Males were significantly taller ( $p < 0.05$ ) had significantly longer ( $p < 0.05$ ) hand and forearm lengths, and had significantly greater hand grip and leg strength ( $p < 0.05$ ). Both sexes showed significant increase ( $p < 0.05$ ) in heart rate, blood lactate with a significant decrease in strength indices from pre to post swim. Significant changes in clean swim speed, stroke index and stroke length ( $p < 0.05$ ) were found between laps. Overall the male participants had significantly ( $p < 0.01$ ) faster swim speeds as a result of significantly longer SL ( $p = .016$ ) with no significant difference in SR ( $p = .064$ ). There was no significant difference ( $p > 0.05$ ) between sexes in the percentage of



time spent in either CPhase 1 or CPhase 2. There was no significant difference in the time spent in any of the four phases of the stroke between sexes ( $p < 0.05$ ). The sex differences in CSS can be attributed to significantly ( $p < 0.05$ ) longer forearm and hand length which increases propelling surface and produces longer SL. The strength deficit and high levels of blood lactate post swim indicate that as participants progressed through the swim they became fatigued which resulted in a decrease in their CSS but did not result in changes to the timing of the stroke indicating a loss of stroke efficiency.

## 2.2 Introduction

Skilled breaststroke swimming is inherently an in-phase rhythmical movement that involves stable and flexible modes of co-ordination between the arms and the legs. These movements arise as a result of the interactions between the mechanical properties of the water and the intrinsic dynamics of the body (Seifert, Chollet and Bardy, 2004). In competitive breaststroke swimming three styles of co-ordination have been observed; 1) *Glide*, which is characterised by a glide phase following the end of the arm recovery phase prior to the start of the leg kick; 2) *Continuous*, which is characterised by the start of the leg kick following the end of the arm recovery phase; 3) *Overlap*, which is characterised by the initiation of the leg kick before the completion of the arm recovery phase (Maglischo, 2003). The investigation of co-ordination changes during a race could provide a better understanding of the swimmer's personal co-ordination style, and how modifications in co-ordination relate to stroke length, stroke rate and swimming speed.

Swimming speed is the result of an inverse relationship between the stroke length (distance per cycle) and the stroke rate (a number of cycles per unit of time). Swimming speed is increased with an increase in stroke rate while stroke length remains constant for lowest speed and decreases as the speed continues to increase (Sidney et al., 2011). Therefore the swimmer must find the optimal combination of stroke length and stroke rate to reach and maintain the highest speed possible within the constraints of the task (Vereijken and Whiting, 1990). In breaststroke swimming it has been showed that as swimmers progress through a competitive 100 m swim there is a significant decrease in the swimmers speed due to significant decreases in both stroke rate and stroke length (Thompson, Haljand and MacLaren, 2000).

Male breaststroke swimmers have been shown to have higher linear velocities resulting from longer stroke lengths and higher stroke rates than females (Seifert and Chollet, 2005). The differences between male and female swimmers in the combinations of stroke rate and stroke length and arm and leg co-ordination may be explained by anthropometric differences such as arm height and arm lengths. However such differences in stroke characteristics may also be dependent on the swimmer technique and the resultant active drag, linear velocity and the ratio of stroke length and stroke rate (Kolmogorov and Duplischeva, 1992). Therefore, differences in the ratio of stroke rate and stroke length may also be related to the stroke phases; arm and leg recovery and propulsive phases along with the glide phase (Chollet et al., 1996; Chollet, Tourny-Chollet and Geizes, 1999; Soares, Sousa and Vilas-Boas, 1999; Seifert and Chollet, 2005). The underwater recovery phases of the breaststroke and the glide phase are characterised by a decrease in linear velocity (D'Acquisto and Costill, 1988; Leblanc et al., 2007) and should be performed in the most streamlined of positions to limit active drag and to maintain a high linear velocity and stroke length (Kolmogorov et al., 1997).

Male and female breaststroke swimmers organise their arms and legs differently throughout, 50-200 m swims (Seifert and Chollet, 2005). There is currently no information on whether males and females make the similar changes in their phasing of the arms and legs as they progress through a race. As pace increases from 200 to 50 m there is an increase in the propulsive phase and a reduction in the glide phases of the stroke cycle in males and females (Seifert and Chollet, 2005). The spatio-temporal differences between males and females may be due to anthropometric differences and different stroke phase durations linked to arm – leg co-ordination (Seifert and Chollet, 2005). It has previously been shown that a difference in anthropometry between the sexes mediates differences in stroke length, stroke rate and

velocity in front crawl swimming (Chatard, Lavoie and Lacour, 1991; Grimston and Hay, 1986). Male swimmers have been reported to have greater stature (height) and longer segment lengths which have also been linked to greater propelling efficiency and longer stroke lengths in front crawl swimming (Toussaint, Janssen and Kluft, 1991; Kjendlie, Stallman and Stray-Gundersen, 2004). Stroke index (SI) as defined by Costill et al., (1985) as the product of average velocity ( $v$ ) and stroke length (SL) is considered a valid indicator of swimming efficiency. Female swimmers are reported to be more efficient than their male counterparts in breaststroke swimming due to the elevated position they adopt in the water. This is a result of better buoyancy in females due to greater percentage body fat in the thighs than males (McLean and Hinrichs, 1998) resulting in a higher hip position. This higher hip position reduces the active drag by reducing the cross sectional area exposed to the water's flow (Leblanc, Seifert and Chollet, 2010). The effect of the increased buoyancy in female swimmers seems to be of less importance during sprint events as the buoyancy effect is only an advantage during a certain range of velocity (Leblanc, Seifert and Chollet, 2010). This is due to the fact that in sprint events the swimmers have a higher linear velocity which causes the legs to be raised as a result of the increased hydrostatic lift forces (Miyashita and Tsunoda, 1978; Kjendlie, Stallman and Stray-Gundersen, 2004). What is unclear is how stroke efficiency changes during a swim and whether it differs between males and females.

Using stroke length and stroke rate to explain characteristic differences in swimming performance could obscure the fact that it is the swimmers ability to generate propulsive forces through the effective application of force (Changalar and Brown, 1992; Maglischo, 2003). Swimmers also have to endure high levels of water resistance (Pelayo et al., 2007) which will also affect the swimmers ability to generate power and therefore reduces swimming speed. This is especially true in breaststroke swimming where there are large

fluctuations in linear speed due to phases of negative acceleration as a result of large drag components of forwards movement (Kolmogorov et al., 1997).

There are a number of studies that have examined at the co-ordination of the arms and legs in breaststroke swimming (Chollet, et al., 2004; Leblanc, et al., 2005; Leblanc, et al., 2009; Seifert & Chollet, 2005; Seifert & Chollet, 2009; Soares, et al., 1999) however these studies have all used discontinuous graded protocols of 25 m. These authors have assumed that the different speeds adopted by the swimmers corresponded to the speeds used during 50 to 200 m events. Arm-leg co-ordination has been determined (Chollet, Tourny-Chollet and Geizes, 1999; Leblanc et al., 2005; Seifert and Chollet, 2005) by the measurement of time gaps between the different phases of the arms and legs. These time gaps included  $T1_a$  the time between the end of leg propulsion (considered as the time between the beginning of the backwards movement of the feet – with the legs maximally flexed at the beginning and leg extension) and the beginning of arm propulsion (identified as time between the beginning and end of the backwards movement of the hand) (Seifert and Chollet, 2005). A study by Takagi, et al., (2004) investigated the differences in arm-leg co-ordination in 100 and 200 m competitive breaststroke swimmers during the 9<sup>th</sup> FINA World Swimming Championships Fukuoka 2001. These authors used three measures of arm-leg co-ordination expressed as a percentage of stroke cycle time; 1) Percentage of simultaneous arm and leg recovery (%SRT) which represented a non-propulsive phase in the stroke, 2) Percentage of simultaneous arm and leg propulsion (%SPT) and 3) Percentage arm lag time (%ALT) which represented the time between the start of leg propulsion and the start of the arm propulsion phase. Takagi, *et al.* (2004) reported similar finding to the other studies in breaststroke arm-leg co-ordination (Chollet et al., 2004; Leblanc et al., 2005; Seifert and Chollet, 2005; Soares, Sousa and Vilas-Boas, 1999) that the glide phase (%SPT) decreased with race distance decreased from 200 to

50 m. Takagi, et al. (2004) reported that males had longer %SRT than females over the three race distances 50, 100 and 200 m. Males also reported significantly ( $p < 0.05$ ) longer stroke lengths and higher swim speeds over the same race distances which is supported by previous work (Changlari and Brown, 1992; Kennedy et al., 1990). Takagi, et al., (2004) also reported that stroke rate increased and stroke length decreased significantly ( $p < 0.05$ ) as event distance increased which again agrees with previous studies (Chollet et al., 2004; Leblanc et al., 2005; Seifert and Chollet, 2005; Soares, Sousa and Vilas-Boas, 1999).

A swimmer's speed is directly affected by the athlete's mechanical output (Toussaint et al., 1988). In many types of sprint events athletes' undergo muscular fatigue as the sprint progresses i.e. there is a decrease in the ability to maintain effective mechanical output (Beelen and Sargeant, 1991). Dry land measurements of strength have been reported to be linked to swimming performance (Vorontsov, 2011) but Kjendlie & Stallman, (2011), reported that dry land strength should be related to clean swim speed and not to overall performance due to starting, turning and finishing technique. Dry land strength has been assessed in a number of ways (Miyashita and Kanehisa, 1983; Sharp, Troup and Costill, 1982; Strass, 1988) . One way of assessing dry land strength in relation to swimming performance has been the use of a hand grip dynamometer (Zampagni et al., 2008) which has been able to predict 84 % of performance time variation using age, height and handgrip strength in master's front crawl sprint swimming. A previous study (Colman et al., 2005) of breaststroke assessed dry land strength through a number of isometric contractions of the upper limbs however no assessment was conducted for the lower limbs. As breaststroke uses the legs to a greater extent than the other strokes it is plausible to measure leg strength as a decrease in ability to maintain the force generating capacity is a definition of fatigue (Enoka and Duchateau, 2008) Measurement of leg strength before and after sprint swimming would

potentially enable identification of changes in the muscles' ability to generate force and therefore identify fatigue in a swimmer (Toussaint et al., 2006). A reduction in power-generating capacity has been previously shown in arms only front crawl (Toussaint et al., 2006) from the 1<sup>st</sup> to the 4<sup>th</sup> lap of a 100 m swim using the MAD system. Toussaint, et al., 2006 reported a 13 % decrease in power generating capacity of the arms resulted in a similar 12.4 % decrease in clean swim speed and subsequent changes in stroke rate and stroke length.

A greater understanding of the co-ordination changes that take place during a breaststroke swim and the related changes in stroke rate and stroke length and the relationship with fatigue would give sports scientists, coaches and swimmers an improved understanding of swimming performance. This information could help with the design of interventions (Pelayo et al., 2007) to maximise swim performance during training and in competition settings. It remain unclear in breaststroke swimming if a decrease in swim speed, and the subsequent changes in stroke rate and stroke length, as seen during a race leads to changes in arm and leg co-ordination. Furthermore whether the potential changes seen are the same for males and females has yet to be reported. Co-ordination patterns give valuable information to the swimmer's coach but no information thus far is available on how or if arm and leg co-ordination changes during a race.

### *2.2.1 Aims*

The aims of this study were to: (1) monitor arm-leg co-ordination changes between each lap of a 100 m swim and relate these to changes in clean swim speed, stroke length stroke rate and stroke index. (2) compare arm-leg co-ordination, clean swim speed, stroke rate, stroke length and stroke index between males and females (3.) determine the intra lap variability in arm-leg co-ordination, clean swim speed, stroke rate, stroke length and stroke

index. (4) Evaluate the inter-relationships between swimming kinematics, dry land strength and anthropometry.

### *2.2.3 Hypothesis*

It was hypothesised that there will be a decrease in clean swim speed from the 1<sup>st</sup> to the 4<sup>th</sup> lap with an associated decrease in stroke length and stroke rate as a result of fatigue.

It was hypothesised that males will have higher clean swim speeds and longer stroke lengths than females due to strength and anthropometry differences.

It was hypothesised that there will be a difference in the co-ordination of the arms and legs between sexes.

It was hypothesised that there will be a change in the co-ordination of the arms and legs from the 1<sup>st</sup> to the 4<sup>th</sup> lap.



## **2.3 Materials and Methods**

### *2.3.1 Participants*

Twenty six specialist breaststroke participants (8 female and 18 male) (average: age 18.4 years  $\pm$  2.5) (powered to 64 %) volunteered to participate in this study. The selection criterion of the study was that participants had to be competitive at County standard or above within that season in the 100 m breaststroke event, FINA points  $567 \pm 110$  based on FINA points scoring 2008 for 100 m short course (Splash; FINA point calculator; GeoLogix, Bern, Switzerland). The study was approved by Coventry University's Ethics Committee. The procedures and demands of the study were explained to participants and each participant provided written informed consent.

Anthropometric measures; stature (height to the nearest 0.01 m, Seca stadiometer), body mass (to the nearest 0.1 kg), upper arm length (length from acromiale to radiale to the nearest 0.01 m), forearm length (length from radiale to stylium to the nearest 0.01 m), hand length (length from midstylium to dactylium to the nearest 0.01 m), and arm span (distance between dactyliums of the right and left hand when chest and palms are placed against a wall and the out stretched arms were abducted to the horizontal to the nearest 0.01 m) were made following the International Society for the Advancement of Kinanthropometry (ISAK) protocol (Norton et al., 1996 (Lindsey Carter and Ackland, 1994; Norton et al., 1996) (Table 2.1).

Before filming each swim, the skin overlaying the joint centres (lateral malleolus, lateral femoral condyle, greater femoral trochanter, styloid process, epicondyle of humerus and acromion process) were marked on both sides of the body using black PVC electrical tape (19

mm in width) to help identify their location. Joint centres were marked on both sides of the body as the right side of the body was used for qualitative analysis (Dartfish Trainer 2.5.2.19, Fribourg, Switzerland) on laps 1 and 3 and the left side of the body was analysed on laps 2 and 4. It was assumed that the stroke was symmetrical (Maglischo, 2003).

## 2.3.2 *Isometric Strength Testing*

### 2.3.2.1 *Hand Grip*

The maximal handgrip strength of the left and right hand was measured using a grip strength dynamometer (Lafayette 7801, Lafayette, NI, USA). The grip range was adjusted such that the proximal interphalangeal joint of the index finger was flexed at 90 degrees. Measurements were made with the subjects standing with the elbow comfortably straight and the wrist in mid-pronation and the arm in full extension above the shoulder. The subjects were instructed to maximally grip the dynamometer on each effort, whilst rotating the arm about an anterior-posterior axis to the diametrically opposite position. No involvement of the non-exercising arm was allowed. Three consecutive measures of grip strength were obtained for each hand with a one-minute interval between measures. This rest period has previously been shown to result in less fatigue (7%) than 15 and 30 seconds rest periods (10 and 12% respectively) (Trossman and Li, 1989). The dial of the dynamometer indicated hand grip strength in kilograms. The maximal grip strength of the three trials was calculated in Newtons by multiplying the dynamometer index by  $9.81\text{m}\cdot\text{s}^{-1}$ . The intraclass correlation coefficient (Thomas and Nelson, 2001) was used to determine the repeatability of the three trials for the left hand grip (96 %), right hand grip (97 %) at rest.

#### 2.3.2.2 *Leg Strength*

The maximal leg strength was measured using a leg dynamometer (Takei, model T.K.K. 5402, Tokyo, Japan). Participants were required to stand unshod on the platform of the dynamometer with the trunk erect and the knees flexed at an angle of approximately 135 degrees (Coldwells, Atkinson and Reilly, 1994). Knee angle was set by placing a goniometer (Bodycare Southam, England) on the fulcrum (lateral epicondyle of the femur); the stable arm was set parallel to the lateral midline of the femur and the movement arm was set parallel to the lateral midline of the fibula. The participants reached down with elbows fully extended and gripped the pull bar and the chain length was adjusted appropriately (Coldwells, Atkinson and Reilly, 1994). The participants were instructed to slowly exert as much force on the handle as possible while extending the knees, without using the back muscles, over a period of 10 seconds. The peak force was recorded. Before the swim three consecutive measures of leg strength were obtained with a one-minute interval between measures (Trossman and Li, 1989) with the highest score of the three pulls being recorded. The intraclass correlation coefficient (Thomas and Nelson, 2001) was used to determine the repeatability of the three trials for the leg dynamometer (97%) at rest.

Immediately following the 100 m swim the participants performed one trial of the isometric leg dynamometer test and one trial of hand grip strength test for each hand to give a maximal value. The trials were not repeated 3 times as this would give the swimmer time to recover from the swim and would give improved performance results. Verbal encouragement was given consistently for each trial pre and post swim.

### 2.3.3 *Physiological Measurements*

Heart rate was measured following 15 minutes of seated rest (Polar Sports Tester, Polar Electro, Kempele, Finland) and 25µl of capillary blood was taken from an earlobe and analysed using Lactate Pro analyser (Arkray 1710, Kyoto, Japan) in accordance with BASES Guidelines (1997) for blood lactate concentration. Heart rate was taken again immediately after the subjects had completed the isometric strength test. Blood lactate concentration was sampled again 5 minutes post the 100 m swim. Ratings of perceived exertion (RPE) using the 6-20 Borg Scales (Borg, 1998) were recorded immediately after the participants left the water.

### 2.3.4 *Swim Trials*

Each participant performed a self-selected 800 m warm-up in a 25 m pool (Thompson et al., 2003). Each participant was then instructed to perform a maximal 100 m swim from a water start with no pre conceived pacing strategies.



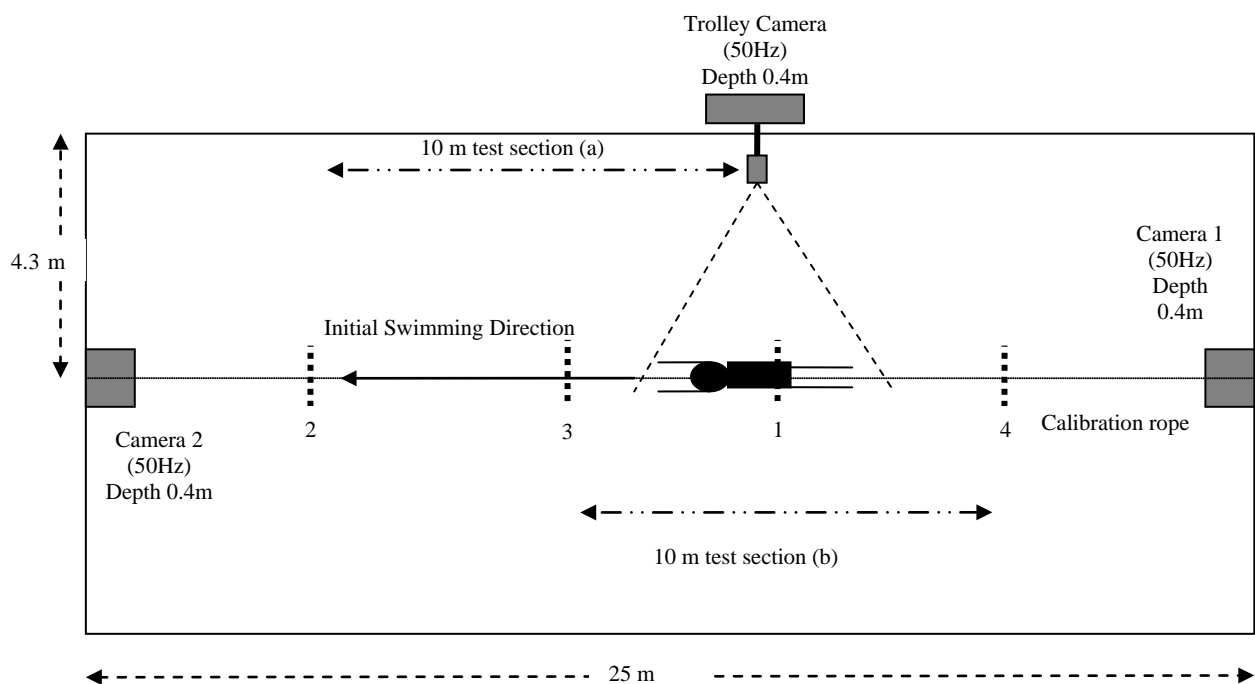
a

b

**Plate 1.0** (a) Front view of the custom made water proof housing for the camcorders made from 8 mm clear Perspex; (b) side view of the custom made water proof housing showing the front aspect of the camcorder was positioned using custom cut foam, to secure it in place, allowing the front aspect of the camcorder to be positioned so that it touched the front panel of the housing.

Three cameras were used to film the 100 m swim. Cameras 1 and 2 (Sony video DCR-TRV460E, Sony, Tokyo, Japan), sampling at 50Hz with the sports preset mode selected. The camera preset was used in sports mode as shutter speed option was not available in camera model. Camera's were enclosed in a custom made waterproof housing (Plate 1.0) at each end of the lane at a depth of 0.4 m, to record frontal and rear views of the swimmer (Figure 2.1). Camera 3 was a waterproof bullet camera, which was suspended underwater from the trolley at a depth of 0.4 m and connected to a visual display unit (VDU) (Sony digital video cassette recorder GV-D800E, Sony, Tokyo, Japan) which was attached to a trolley (Figure 2.1). The field of view was adjusted so that the whole body of the participant was visible (Figure 2.1). The frontal and rear views of the participant were synchronised to the sagittal view (Dartfish Trainer 2.5.2.19, Fribourg, Switzerland) using a custom made LED light trigger system that could be seen in all

camera views. The trolley was manually moved at the same speed as the participants parallel to the greater femoral trochanter. This procedure maintained the joint marker in the approximate centre of the field of view (Figure 2.1), in the sagittal plane, throughout the entire 100 m swim. Refraction due to water was not accounted for in this study as refraction in water due to the light rays has been reported to vary between 0.39 and 1.28 % (Gourgoulis et al., 2008).



**Figure 2.1** Plan view of the filming set-up used for qualitative analysis.

Time to complete 100 m was recorded (to the nearest 0.02s) using a video analysis package (Dartfish Trainer 2.5.2.19, Fribourg, Switzerland) as the time from when the feet left the wall at the start until the double hand touch on the wall at the end of the race.

### 2.3.5 Stroke Parameters

The following stroke parameters were calculated from the 10 m testing sections of the pool on each of the four laps of a 100 m swim from the sagittal view video recordings (trolley camera, Figure 2.1). *Clean Swim speed* ( $\text{m}\cdot\text{s}^{-1}$ ) was defined as the mean forward speed of the greater trochanter over the 10 m testing section on each lap (Figure 2.1) monitored from time the greater trochanter entered the 10 m testing sections until it left the 10 m testing section (Figure 2.1) via the stop clock located on the video analysis package (to the nearest  $0.01 \text{ m}\cdot\text{s}^{-1}$ ); *Stroke frequency* ( $\text{stroke}\cdot\text{min}^{-1}$ ) was defined as the number of stroke cycles performed in one minute, calculated as the mean over each of the 10 m testing sections (Figure 2.1) (to the nearest  $0.01 \text{ strokes}\cdot\text{min}^{-1}$ ); *Stroke length* ( $\text{m}\cdot\text{cycle}^{-1}$ ) was defined as the distance that the participants greater trochanter travelled in one stroke cycle, computed from the clean swim speed and the stroke rate values (to the nearest 0.01m) (Equation 2.1) ; *Stroke Cycle Time* (s) was defined as the time taken to complete one complete stroke cycle, calculated as the mean stroke cycle time over the 10 m testing sections (to the nearest 0.02 s); Stroke index (SI) as defined by Costill et al., (1985) (Equation 2.2) was used to evaluate swimming efficiency. All stroke parameter calculations were taken from the 10 m testing sections of the pool (Figure 2.1) for each of the four laps of the 100 m swim.

$$\text{Equation 2.1 } SL = CSS/SR$$

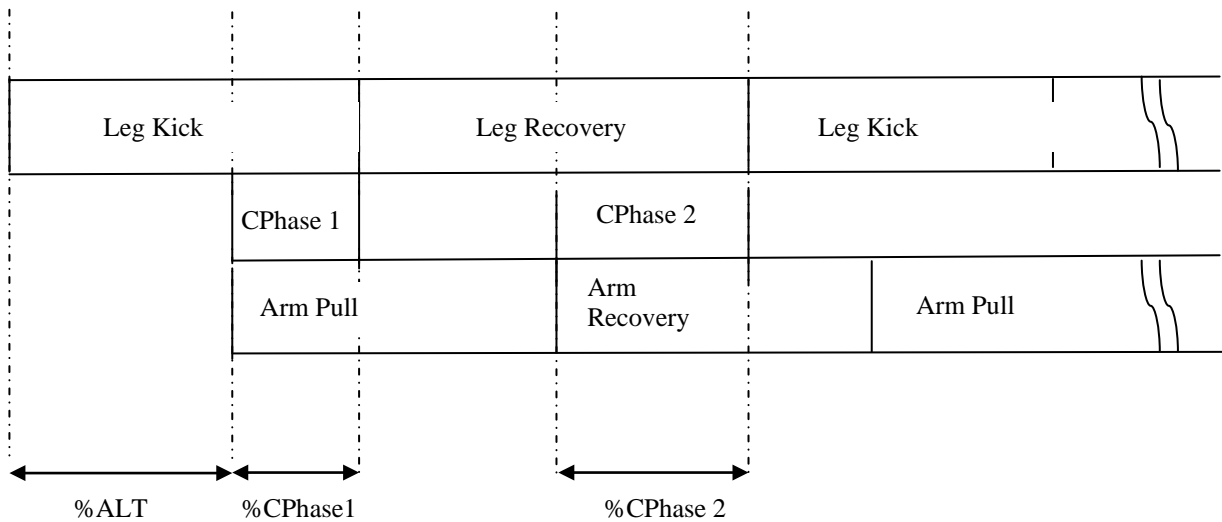
$$\text{Equation 2.2 } SI = V \times SL$$

### 2.3.6 *Arm and Leg Co-ordination and Stroke Phases*

Three complete stroke cycles (Chollet et al., 2004) which were completed within the 10 m testing section of each of the four laps prior to the turn or the finish were analysed using the synchronised frontal and sagittal camera views (Figure 2.1) to determine the average duration of each of the following phases: Arm Pull (the time between the separation of the hands from the extended position in front of the body until the first forward movement of the elbow when the hands were under the head); Arm recovery (the time between the end of the arm pull phase and the start of the separation of the hands from the extended position); Leg kick (the time between the start of the first backwards movement of the feet, the point where the legs were maximally flexed at the start, and the point when the legs were fully extended); Leg recovery (the time between the end of the leg kick phase and the complete flexion of the knee until forward movement of the feet had finished); Co-ordination phase 1 (CPhase1) was calculated as the time between the end of the leg kick phase and the start of the arm recovery phase and was used to classify the participants co-ordination as overlap (represented by a negative value to the nearest 0.02 s indicating simultaneous propulsion of the arms and legs), glide (represented by a positive value to the nearest 0.02 s indicating a delay (glide) in the initiation of the arm pull phase) or continuous; Co-ordination phase 2 (CPhase 2; the time between the end of the arm pull phase and the start of leg kick phase); Arm lag time (ALT; corresponded to the time from the start of the leg kick to the beginning of arm pull). All phases were expressed as a percentage of total cycle time with a precision of 0.02 s (Figure 2.2). It should be noted that the start of the arm pull phase and the end of the arm pull phase as described above does not necessarily correspond the start and end of the propulsive components of the arms' stroke, similarly for the leg kick, the start of the leg kick and the end of the leg kick does not necessarily correspond to the start and end of the leg propulsion (Maglischo, 2003). The key stroke phases of the arms and legs were subjectively



determined by three independent operators using a blind technique i.e. without knowing the results of the other operators. The three independent analyses were then compared with the mean difference of the operators being ( $< 0.04$  to the nearest  $0.02$  s), which was less than the  $0.04$  s which has previously been used to validate key stroke phases (Seifert, Chollet and Chatard, 2007).



**Figure 2.2** Definition of stroke phase and measurement of arm-leg co-ordination in breaststroke swimming. The block diagram describes the phases of the stroke with time increasing along the horizontal axis.

### 2.3.7 Statistical Procedures

Statistical analyses were conducted using SPSS version 16.0 (SPSS, Inc., Chicago, IL, USA). Standard statistical methods were used to calculate mean and standard deviation for all measured variables. Normal distribution of the data was verified using Levenes test. The percentage data was tested for normal distribution using the  $z$ -score of skewness and kurtosis in accordance with Vincent, (2005). If normal distribution was not reported then data was corrected for heterogeneity of variance using the Arcsine transformation (Black, 1999). The level of significance was set at  $p < 0.05$ . Independent T tests were used to determine sex differences within the anthropometry data. The effect size of the independent T test was estimated using Pearson's correlation coefficient in accordance with Rosnow & Rosenthal, (2005), values interpreted according to Cohen, (1988) as  $r = 0.10$  (small effect),  $r = 0.30$  (medium effect) and  $r = 0.50$  (large effect) results and reported in accordance with Field (2009). Two-way Analysis of Variance was used to analyse physiology and strength measures with time (pre and post) and sex (males and females) as the fixed factors. Left and right hand strength performance being analysed in separate ANOVAs. Two-way Analysis of variance was used to compare selected kinematic variables at the same point of each of the four laps, with lap (1, 2, 3 and 4) and Sex (males and female) as the fixed factors. When Mauchley's test of sphericity indicated a minimal level of violation ( $> 0.75$ ) the degrees of freedom was corrected using the Huynh-Feldt adjustment and when the sphericity was  $< 0.75$ , the Greenhouse-Geiser correction was used (Field, 2009). The degrees of freedom were adjusted for the main effect of lap with stroke length and stroke index. Where differences were noted in ANOVA, pairwise comparisons (Bonferroni adjusted) were employed to identify where the significant differences occurred. A level of  $p < 0.05$  was

considered statistically significant. The effect size for the ANOVA statistics was estimated using partial Eta squared ( $\eta p^2$ ) for analysis of variance. Since this method is likely to overestimate effect sizes, values were interpreted according to Ferguson (2009) as no effect if  $0 \leq \eta p^2 < 0.05$ ; a minimum effect if  $0.05 \leq \eta p^2 < 0.26$ ; a moderate effect if  $0.26 \leq \eta p^2 < 0.64$ ; and a strong effect if  $\eta p^2 \geq 0.64$ . All ANOVA results were reported in accordance with Field (2009). Pearson product moment correlation coefficients were determined among selected kinematic variables for each of the four laps. The truncated product method (Zaykin et al., 2002) was used to combine a set of  $p$ -values (those for the Pearson product moment correlations) to determine whether it was likely that all significant results in the set were truly significant or were due to chance.

## 2.4 Results

### 2.4.1 Anthropometric and Physiological Data

The mean values for the anthropometry data (Table 2.1) show a significant difference between men and women for height  $t(24) = 3.13$ ,  $p = .005$ ,  $r = 0.56$ , arm span  $t(24) = 2.52$ ,  $p = .02$ ,  $r = 0.46$ , forearm length  $t(24) = 2.23$ ,  $p = .035$ ,  $r = 0.41$  and hand length  $t(24) = 2.11$ ,  $p = .045$ ,  $r = 0.40$ , strength means are shown in table 2.2. There was a significant main effect of sex for hand grip with the males producing significantly more force for both the left  $F(1,24) = 17.1$ ,  $p = .001$ ,  $\eta p^2 0.42$  and right hand  $F(1,24) = 577.16$ ,  $p = .001$ ,  $\eta p^2 0.96$  pre swim. There was also a significant main effect of sex for leg dynamometry  $F(1,24) = 14.2$ ,  $p = .001$ ,  $\eta p^2 .371$  with the males on average producing 65 % more force than the females pre swim. Both males and females showed a significant decrease in strength scores post the 100 m swim for hand grip of the left  $F(1,24) = 24.20$ ,  $p < .001$ ,  $\eta p^2 0.50$  (Table 2.2) and right

hand  $F(1,24) = 15.05$ ,  $p = .001$ ,  $\eta^2 0.39$ , (Table 2.2 ) and leg strength  $F(1,24) = 7.81$ ,  $p = .01$ ,  $\eta^2 0.25$ . There was no significant main effect of sex for blood lactate  $F(1,24) = 215$ ,  $p = .170$ ,  $\eta^2 0.07$  (Table 2.3) or heart rate  $F(1,24) = 0.172$ ,  $p = .682$ ,  $\eta^2 0.01$  (Table 2.3). There was a significant increase in both heart rate (134 %)  $F(1,24) = 271$ ,  $p < 0.001$ ,  $\eta^2 0.92$  (Table 2.3) and blood lactate concentrations (526 %)  $F(1,24) = 125$ ,  $p < 0.001$ ,  $\eta^2 .839$  (Table 2.3) from before the swim to 5 minutes post the 100 m swim. There was no significant difference  $t(24) = -0.34$ ,  $p = .299$ ,  $r 1.0$  (Table 2.3) in sex for post swim rating of perceived exertion (RPE).

**Table 2.1** Anthropometric measures and performance time of the participants n=26. All values are given as mean  $\pm$ SD.

Participant	Age		Body mass	Arm	Upper Arm	Forearm	Hand	Performance
	(years)	Height (m)		Span	Length	Length	Length	Time 100m
			(kg)	(m)	(m)	(m)	(m)	(s)
Female (n=8)	19.1 $\pm$ 2.3	1.70 $\pm$ 0.05*	69.0 $\pm$ 8.0	1.73 $\pm$ 0.07*	0.33 $\pm$ 0.02	0.24 $\pm$ 0.02*	0.19 $\pm$ 0.01*	88.3 $\pm$ 5.4*
Male (n=18)	18.9 $\pm$ 2.2	1.78 $\pm$ 0.06	69.3 $\pm$ 7.3	1.83 $\pm$ 0.10	0.34 $\pm$ 0.03	0.26 $\pm$ 0.02	0.20 $\pm$ 0.01	77.5 $\pm$ 5.5

\*Denotes statistically significant difference  $p < 0.05$  between sexes

**Table 2.2** Strength measures at rest and post 100 m swim of the participants n=26. All values are given as mean  $\pm$ SD and coefficient variation percentage (CV %).

	Resting Left	Post Swim Left	Resting Right	Post Swim Right		
	Hand Grip	Hand Grip	Hand Grip	Hand Grip	Resting Leg	Post Swim Leg
	Strength	Strength	Strength	Strength	Strength	Strength
Participant	(N)	(N)	(N)	(N)	(N)	(N)
Female (n=8)	311 $\pm$ 74 (cv=24)*	259 $\pm$ 55 (cv=21) <sup>#</sup>	294 $\pm$ 63 (cv=21)*	262 $\pm$ 66 (cv=25) <sup>#</sup>	768 $\pm$ 176 (cv=23)*	728 $\pm$ 197 (cv=27) <sup>#</sup>
Male (n=18)	433 $\pm$ 83 (cv=19)	377 $\pm$ 68 (cv=18) <sup>#</sup>	432 $\pm$ 68 (cv=16) <sup>#</sup>	384 $\pm$ 79 (cv=21) <sup>#</sup>	1268 $\pm$ 331 (cv=26) <sup>#</sup>	1149 $\pm$ 330 (cv=29) <sup>#</sup>

\*Denotes statistically significant difference  $p < 0.05$  between sexes.

<sup>#</sup>Denotes statistically significant difference  $p < 0.05$  between resting and post swim strength values.

**Table 2.3** Physiological measures at rest and post 100 m swim of the participants n=26. All values are given as mean  $\pm$ SD.

	Resting	Post Swim	Resting	Post Swim	Post Swim
	Heart Rate	Heart Rate	Blood lactate	Blood Lactate	RPE
Participant	(Beats $\cdot$ min $^{-1}$ )	(Beats $\cdot$ min $^{-1}$ )	(mmol $\cdot$ L $^{-1}$ )	(mmol $\cdot$ L $^{-1}$ )	
Female (n=8)	79 $\pm$ 11 <sup>#</sup>	183 $\pm$ 10 <sup>#</sup>	1.0 $\pm$ 0.3 <sup>#</sup>	6.6 $\pm$ 2.2 <sup>#</sup>	18 $\pm$ 1
Male (n=18)	75 $\pm$ 17	173 $\pm$ 30	1.3 $\pm$ 0.4	8.0 $\pm$ 2.8	17 $\pm$ 1

<sup>#</sup> Denotes statistically significant difference  $p < 0.05$  sexes

**Table 2.4** Summary of correlations between absolute kinematic dependent variables and anthropometric independent variables

	Height	Mass	Arm Span	Upper Arm Length	Forearm Length	Hand Length
<i>r</i> value for CSS absolute values <sup>a</sup>	.248	.120	.162	-.248	.187	.038
<i>p</i>	.222	.560	.431	.222	.362	.855
<i>r</i> value for SR absolute values <sup>b</sup>	-.314	.000	-.454	-.144	-.347	-.334
<i>p</i>	.118	.999	.020	.482	.083	.095
<i>r</i> value for SL absolute values <sup>c</sup>	.368	.01	.439	-.052	.403	.291
<i>p</i>	.064	.960	.025	.800	.041	.150

The truncated product method calculated *p* values for the repeated correlations of <0.0001 for *r* values for CSS, SR and SL.

*r* values were calculated as Pearson product moment correlations



#### 2.4.2 Performance Data

Analysis of variance of clean swim speed (CSS) showed a significant main effect for sex  $F(1,24) = 5048, p < .001, \eta^2 0.89$  (Table 2.5). On average over the four laps males had significantly higher (8 %) CSS than females. There was a significant main effect for lap  $F(3,72) = 37.31, p < .001, \eta^2 0.61$  (Table 2.5) with post hoc comparisons indicating a significant decrease in CSS from the 1<sup>st</sup> to 2<sup>nd</sup> ( $p = .006$ ), 2<sup>nd</sup> to 3<sup>rd</sup> laps ( $p < .001$ ) with an overall significant ( $p < .001$ ) decrease in CSS of 9 % from the 1<sup>st</sup> to the 4<sup>th</sup> Lap. There was no significant ( $p = .367$ ) change in CSS from the 3<sup>rd</sup> to the 4<sup>th</sup> lap. There was no significant interaction in CSS between sex and lap  $F(3,72) = 1.64, p = .187, \eta^2 0.06$ , (Table 2.5) indicating that males and females were following the same trend with a decrease in CSS from the 1<sup>st</sup> to 4<sup>th</sup> Lap. The mean CSS over the 4 laps showed significant correlations with pre swim hand grip strength for both left ( $r = .446, p < 0.05$ ) and right hand ( $r = 0.41, p < 0.05$ ) but not for leg strength.

Stroke length (SL) showed a significant main effect for sex  $F(1,24) = 6.711, p = .016, \eta^2 0.22$  (Table 2.5) with the males on average having a 15 % longer SL. There was a significant main effect for lap  $F(2.6,62.4) = 4.79, p = .007, \eta^2 0.17$  (Table 2.5) with post hoc comparisons showing a significant decrease only between the 2<sup>nd</sup> and 4<sup>th</sup> lap of the swim. There was no significant interaction for SL between sex and lap  $F(1,24) = .129, p = .723, \eta^2 0.01$  (Table 2.5). The mean SL over the four laps showed significant correlation with average CSS ( $r = .540, p < 0.01$ ). The mean SL over the four laps also showed significant correlation with forearm length ( $r = .397, p < 0.05$ ) and a significant negative correlation with arm span ( $r = -.454, p < 0.05$ ). The truncated product moments for correlations of the kinematic data and anthropometric variables can be found in Table 2.4.

For stroke rate (SR) there was no significant main effect for sex  $F(1,24) = 1.18, p = .287, \eta^2 0.06$  (Table 2.5). There was a significant main effect for lap  $F(3,72) = 4.14, p = .009, \eta^2 0.15$  (Table 2.5) with post hoc comparisons indicating a significant decrease in SR from the 1<sup>st</sup> to 2<sup>nd</sup> lap ( $p = .016$ ) and from the 1<sup>st</sup> to 3<sup>rd</sup> lap ( $p = .044$ ) whereas there was no significant change from the 1<sup>st</sup> to the 4<sup>th</sup> lap ( $p = .262$ ). There was no significant interaction for SR between sex and lap indicating that males and females are changing SR in the same pattern over the swim.

Stroke Index (SI) showed a significant main effect for sex  $F(1,24) = 618.7, p = .003, \eta^2 0.31$  (Table 2.5) with males on average having a 31 % higher SI than females. There was a significant main effect for lap  $F(2.1,49.3) = 14.4, p < .001, \eta^2 0.38$  (Table 2.5) with post hoc comparisons showing a significant decrease in SI from the 1<sup>st</sup> to 3<sup>rd</sup> ( $p = .012$ ) and 1<sup>st</sup> to 4<sup>th</sup> lap ( $p < 0.001$ ). There was no significant interaction between sex and lap for SI.

**Table 2.5** Mean  $\pm$  SD values and coefficient of variation percentage (CV %) for Clean swim speed (CSS), stroke length (SL), stroke rate (SR), stroke cycle time and stroke index (SI) for males (n=18) and females (n=8) over the four laps of the 100 m swim

	1st Lap	2nd Lap	3rd Lap	4th Lap
CSS (m·s <sup>-1</sup> )				
Male <sup>a</sup>	1.24 $\pm$ 0.10 (cv=8)	1.19 $\pm$ 0.07 (cv=6.2)	1.13 $\pm$ 0.07 (cv=6.5)	1.14 $\pm$ 0.08 (cv=7)
Female	1.11 $\pm$ 0.06 (cv=5.5)	1.07 $\pm$ 0.08 (cv=7.5)	1.04 $\pm$ 0.08 (cv=7.5)	1.00 $\pm$ 0.08 (cv=7.9)
<b>Group Mean</b>	<b>1.20<math>\pm</math>0.11</b>	<b>1.15<math>\pm</math>0.09<sup>b</sup></b>	<b>1.10<math>\pm</math>0.08<sup>c,e</sup></b>	<b>1.10<math>\pm</math>0.10<sup>d</sup></b>
SL (m·cycle <sup>-1</sup> )				
Male <sup>a</sup>	1.62 $\pm$ 0.24 (cv=15)	1.64 $\pm$ 0.22 (cv=13.7)	1.57 $\pm$ 0.22 (cv=13.9)	1.55 $\pm$ 0.24 (cv=15.4)
Female	1.39 $\pm$ 0.24 (cv=17)	1.39 $\pm$ 0.24 (cv=17)	1.36 $\pm$ 0.27 (cv=19.7)	1.28 $\pm$ 0.22 (cv=17.1)
<b>Group Mean</b>	<b>1.55<math>\pm</math>0.26</b>	<b>1.56<math>\pm</math>0.25</b>	<b>1.50<math>\pm</math>0.25</b>	<b>1.47<math>\pm</math>0.26</b>
SR (stroke·min <sup>-1</sup> )				
Male	46.8 $\pm$ 7.4 (cv=15.8)	44.3 $\pm$ 6.5 (cv=14.7)	43.7 $\pm$ 5.6 (cv=12.7)	44.8 $\pm$ 6.0 (cv=13.4)
Female	49.7 $\pm$ 8.2 (cv=16.6)	47.2 $\pm$ 6.8 (cv=14.4)	47.2 $\pm$ 8.4 (cv=17.7)	47.3 $\pm$ 7.7 (cv=16.2)
<b>Group Mean</b>	<b>43.3<math>\pm</math>6.8</b>	<b>41.3<math>\pm</math>6.4<sup>b</sup></b>	<b>41.2<math>\pm</math>6.8<sup>c</sup></b>	<b>42.0<math>\pm</math>6.3</b>
Stroke Cycle time (s)				
Male	1.45 $\pm$ 0.22 (cv=15.3)	1.52 $\pm$ 0.22 (cv=14.5)	1.53 $\pm$ 0.21 (cv=13.4)	1.49 $\pm$ 0.20 (cv=13.2)
Female	1.36 $\pm$ 0.26 (cv=18.9)	1.40 $\pm$ 0.26 (cv=18.2)	1.40 $\pm$ 0.28 (cv=19.9)	1.38 $\pm$ 0.25 (cv=18.2)
<b>Group Mean</b>	<b>1.42<math>\pm</math>0.23</b>	<b>1.48<math>\pm</math>0.23</b>	<b>1.49<math>\pm</math>0.23</b>	<b>1.46<math>\pm</math>0.22</b>
Stroke Index (m <sup>2</sup> ·s <sup>-1</sup> )				
Male <sup>a</sup>	2.01 $\pm$ 0.41 (cv=20.2)	1.95 $\pm$ 0.32 (cv=16.3)	1.77 $\pm$ 0.32 (cv=17.8)	1.77 $\pm$ 0.36 (cv=20.2)
Female <sup>a</sup>	1.54 $\pm$ 0.26 (cv=16.9)	1.49 $\pm$ 0.32 (cv=21.7)	1.41 $\pm$ 0.33 (cv=23.5)	1.29 $\pm$ 0.26 (cv=20.4)
<b>Group Mean</b>	<b>1.87<math>\pm</math>0.42</b>	<b>1.81<math>\pm</math>0.38</b>	<b>1.66<math>\pm</math>0.36<sup>c,e</sup></b>	<b>1.62<math>\pm</math>0.40<sup>d</sup></b>

<sup>a</sup> Denotes a statistically significant difference  $p < 0.05$  between the sexes.

<sup>b</sup> Denotes a statistically significant difference  $p < 0.05$  between the 1<sup>st</sup> and 2<sup>nd</sup> lap

<sup>c</sup> Denotes a statistically significant difference  $p < 0.05$  between the 1<sup>st</sup> and 3<sup>rd</sup> lap

<sup>d</sup> Denotes a statistically significant difference  $p < 0.05$  between the 1<sup>st</sup> and 4<sup>th</sup> lap

<sup>e</sup> Denotes a statistically significant difference  $p < 0.05$  between the 2<sup>nd</sup> and 3<sup>rd</sup> lap

<sup>f</sup> Denotes a statistically significant difference  $p < 0.05$  between the 2<sup>nd</sup> and 4<sup>th</sup> lap

### 2.4.3 *Arm-Leg Co-ordination*

Nine participants (four females and five males) utilised the overlap co-ordination technique, thirteen participants (three females and ten males) utilised glide co-ordination technique and four participants (one female and three males) started with the glide co-ordination technique but changed to the overlap co-ordination between the 1<sup>st</sup> and the 4<sup>th</sup> lap. Of the four participants that changed from the glide to the overlap co-ordination technique, three participants (one female and two males) altered their co-ordination on the final lap and the other participant (one male) changed their co-ordination technique on the 2<sup>nd</sup> lap.

For all the arm and leg phases of the stroke cycle (Table 2.6) there was no significant main effect for lap ( $p > 0.05$ ) or sex ( $p > 0.05$ ) and there were also no significant interaction between sex and lap ( $p > 0.05$ ). For the arm-leg co-ordination phases CPhase 1, CPhase 2 and ALT there were no significant main effects or interactions for lap and sex ( $p > 0.05$ , Table 2.6)

**Table 2.6** Mean  $\pm$  SD values and coefficient of variation percentage (CV %) of arm and leg stroke phases and arm-leg co-ordination expressed as a percentage for males (n=18) and females (n=8) over the four laps of the 100 m swim

	1st Lap	2nd Lap	3rd Lap	4th Lap
Leg Kick (%)				
Male	21.6 $\pm$ 5.4 (cv=23.4)	23.0 $\pm$ 6.8 (cv=29.6)	22.9 $\pm$ 6.4 (cv=27.9)	24.0 $\pm$ 8.3 (cv=34.7)
Female	21.6 $\pm$ 7.0 (cv=31.2)	22.0 $\pm$ 8.2 (cv=36.4)	21.0 $\pm$ 7.5 (cv=34.9)	22.7 $\pm$ 8.3 (cv=36.6)
Leg Recovery (%)				
Male	76.8 $\pm$ 5.4 (cv=7.1)	77.0 $\pm$ 6.8 (cv=8.8)	77.1 $\pm$ 6.4 (cv=8.3)	76.0 $\pm$ 8.3 (cv=11)
Female	74.6 $\pm$ 5.0 (cv=6.7)	73.9 $\pm$ 7.6 (cv=10.2)	75.1 $\pm$ 6.6 (cv=8.8)	73.1 $\pm$ 7.9 (cv=10.7)
Arm Pull (%)				
Male	46.1 $\pm$ 8.1 (cv=17.6)	46.0 $\pm$ 8.8 (cv=19.1)	45.5 $\pm$ 8.4 (cv=18.4)	47.7 $\pm$ 8.6 (cv=18.1)
Female	48.1 $\pm$ 10.3 (cv=21.3)	48.5 $\pm$ 10.4 (cv=21.5)	48.4 $\pm$ 9.4 (cv=19.4)	47.7 $\pm$ 7.8 (cv=16.4)
Arm Recovery (%)				
Male	53.9 $\pm$ 8.1 (cv=15)	54 $\pm$ 8.8 (cv=16.3)	54.5 $\pm$ 8.4 (cv=15.4)	52.3 $\pm$ 8.6 (cv=16.5)
Females	51.9 $\pm$ 10.3 (cv=19.8)	51.5 $\pm$ 10.4 (cv=20.3)	51.6 $\pm$ 9.4 (cv=18.2)	52.3 $\pm$ 7.8 (cv=15)
Co-ordination Phase 1(%)				
Male	7.3 $\pm$ 18.6	5.9 $\pm$ 16.9	5.5 $\pm$ 16.1	0.6 $\pm$ 18
Female	-0.4 $\pm$ 13.3	-1.4 $\pm$ 13.7	-0.4 $\pm$ 12.3	-2.9 $\pm$ 15.5
Co-ordination Phase 2 (%)				
Male	23.4 $\pm$ 9.7 (cv=41.4)	25.3 $\pm$ 4.7 (cv=18.7)	26.3 $\pm$ 5.4 (cv=20.7)	27.7 $\pm$ 5.2 (cv=18.7)
Female	27.1 $\pm$ 6.5 (cv=24)	26.7 $\pm$ 4.8 (cv=26.1)	27.2 $\pm$ 4.8 (cv=17.7)	28.3 $\pm$ 5.6 (cv=19.9)
ALT (%)				
Male	30.7 $\pm$ 14.5 (cv=47.1)	28.9 $\pm$ 11.5 (cv=39.6)	28.4 $\pm$ 10.9 (cv=38.2)	24.6 $\pm$ 11.4 (cv=46.6)
Female	25.3 $\pm$ 8.7 (cv=34.3)	24.7 $\pm$ 6.6 (cv=26.7)	24.5 $\pm$ 6.9 (cv=28.1)	24 $\pm$ 8.2 (cv=34.2)

**Table 2.7** Summary of correlations between absolute kinematic independent variables

	<b>CPhase 1</b>	<b>CPhase 2</b>	<b>%ALT</b>	<b>%Arm Pull</b>	<b>% Arm Recovery</b>	<b>% Leg Kick</b>	<b>% Kick Recovery</b>
<i>r</i> value for CSS absolute values <sup>a</sup>	-.066	-.203	.014	.089	-.089	.192	-.191
<i>p</i>	.748	.320	.947	.666	.666	.348	.350
<i>r</i> value for SR absolute values <sup>b</sup>	-.501	.025	-.522	.618	-.618	.405	-.405
<i>p</i>	.009	.903	.006	.001	.001	.040	.040
<i>r</i> value for SL absolute values <sup>c</sup>	.325	-.092	.382	-.415	.415	-.191	.191
<i>p</i>	.106	.656	.054	.035	.035	.351	.350

The truncated product method calculated an overall *p* value of <0.0001 for the data given in this table.

<sup>a</sup> Individual Pearson product moment correlations between clean swim speed (CSS) and kinematic independent variables.

<sup>b</sup> Individual Pearson product moment correlations between stroke rate (SR) and kinematic independent variables.

<sup>c</sup> Individual Pearson product moment correlations between stroke length (SL) and kinematic independent variables.

## 2.5 Discussion

### 2.5.1 Stroke Parameters

The findings of the current study show that there was a significant decrease ( $p < 0.05$ , Table 2.5) in the participants clean swim speed (CSS) over the duration of the 100 m swim. The highest CSS was reported on the 1<sup>st</sup> lap of the swim with a decrease of 8 and 9 % respectively through to the 3<sup>rd</sup> and 4<sup>th</sup> laps of the swim. This drop off in CSS was similar to the 7 % reported by Thompson, et al., (2000) from the 1<sup>st</sup> to 2<sup>nd</sup> lap of a 100 m long course breaststroke swim. The decrease in CSS swim speed was related to the change in the ratio of stroke length (SL) and stroke rate (SR) as there was a significant decrease ( $p < 0.05$ ) in SR from the 1<sup>st</sup> to the 2<sup>nd</sup> and 3<sup>rd</sup> laps of the swim with an increase seen on the final lap as there was no significant difference to the 1<sup>st</sup> lap. There was no significant change in SL over the duration of the swim. This is in contrast to finding of a previous study (Thompson, Haljand and MacLaren, 2000) that reported a significant decrease of 9.7 % decrease in SL which was attributed to swimming the 1<sup>st</sup> lap significantly faster and swimmers being unable to maintain their SL. The participants in the current study used positively paced swimming strategies similar to those reported during competition (Thompson, et al., 2000) even though they were asked not to use preconceived race strategies.

The change in CSS over the duration of a breaststroke swim has been suggested to occur as a result of the onset of fatigue in the leg muscles due to the heavy reliance on the legs for propulsion in breaststroke swimming (Maglischo, 2003) resulting in metabolic acidosis (Thompson, 1998). Fatigue is a term used to denote a transient decrease in the capacity to perform physical activity (Enoka and Duchateau, 2008), as shown in the current study by the decrease in CSS over the duration of the 100 m swim. The present study also demonstrated a ( $p$

< 0.05, Table 2.2) decrease of 10 % in the force produced by the leg muscles on the leg dynamometer and a similar ( $p < 0.05$ , Table 2.2) reduction in hand grip dynamometer performance of 11 and 13 % in the left and right hand respectfully. One of the reasons for a decrease in force production could be due to local muscle fatigue connected to metabolic acidosis as shown in the current study by the high levels of blood lactate recorded after the 100 m swim (Table 2.3). There could also be an unmeasured component of central fatigue in the present study, leading to an inhibition of the working muscles as a result of afferent feedback from the muscles, joints and tendons inhibiting motor activity at the spinal or supraspinal levels contributing to the observed loss of swimming performance (James, et al., 1995). It has been previously reported that strength should be investigated in relation to CSS (Kjendlie and Stallman, 2011) and not overall performance time. The hand grip values of the left and right hand showed significant correlations with CSS however, the leg dynamometer results did not. This may be due to a learning effect that has been shown to occur with the leg dynamometer such that more practice is required to achieve maximal values (Coldwells, Atkinson and Reilly, 1994). This indicates that the leg dynamometer may not have provided a true indication of the participant's leg strength, but it may also be due to the fact that swimming performance is as dependant on optimal technique and minimal resistive forces than it is on muscular strength (Kolmogorov et al., 1997; Tanaka et al., 1993).

The decrease in the participant's ability to maintain force application from the 1<sup>st</sup> to the 4<sup>th</sup> lap may have resulted in a decrease in the force generated during the propulsive phases of the stroke cycle thus resulting in an overall decrease in CSS. This is further supported by the decrease in Stroke Index (SI) over the duration of the swim. The SI indicates that the participants are becoming less efficient as they progress through the swim as a result of a decrease in SL and the subsequent decrease in CSS resulting in poor body alignment and increased drag. The



decrease in SI due to fatigue mechanisms could lead the participants to utilise compensatory mechanisms to try and maintain CSS. It has been previously reported that compensatory mechanisms (Forestier and Nougier, 1998) of fatigue are such that other muscles take over the function of the muscles that normally perform the repetitive task. Further studies are needed to investigate these compensatory mechanisms and see how a change in muscle activation affects the mechanical efficiency of swimmers.

### 2.5.2 Sex Effect

The overall results of the current study show that males had significantly (by 8 %,  $p < 0.05$ , Table 2.5) higher CSS than females over the four laps which is consistent with previous studies (Seifert and Chollet, 2005; Takagi et al., 2004). The higher CSS of the male swimmers could be due to the fact that males had on average a 15 % ( $p < 0.05$ ) longer SL than females however both reported similar SR (Table 2.5) which results in the males being able to achieve greater CSS as velocity is a product of SL and SR. This finding of males achieving significantly ( $p < 0.05$ ) higher CSS and longer SL is consistent with previous literature (Changlari and Brown, 1992; Thompson, Haljand and MacLaren, 2000; Takagi et al., 2004). Longer SL in males have been attributed to the fact that males were 4.7 % ( $p < 0.05$ , Table 2.1) taller than females and also presented significantly ( $p < 0.05$ ) longer segment lengths for the hand (5.6 %) and forearm (8 %). The longer segment lengths and greater height have been strongly correlated to SL in front crawl swimming (Chatard, Lavoie and Lacour, 1991; Grimston and Hay, 1986). It has been reported that males (Seifert and Chollet, 2005) have a greater capacity to overcome active drag so are able to swim faster. The greater segment lengths of the male participants have previously been linked to greater propelling efficiency (Toussaint, Janssen and Kluft, 1991; Kjendlie, Stallman and Stray-Gundersen, 2004). The lower propelling efficiency of the female

participants could be due to the fact females have previously been reported to have lower active drag values ( $D = 24 v^2$ ) than males ( $D = 30 v^2$ ) in front crawl swimming (Toussaint et al., 1988). The previously reported lower cross-sectional area ( $0.0075 \text{ m}^2$  vs.  $0.091 \text{ m}^2$ ) of females along with smaller hand and foot lengths produces lower active drag at comparable velocities (Toussaint et al., 1988). Females also have lower active drag values due to a more horizontal position which also reduces the active hydrodynamic coefficient (Kolmogorov et al., 1997). Male swimmers have also been reported to generate greater mechanical power outputs ( $P_d$ ) than females (Kolmogorov et al., 1997). This can be explained by the increased propelling surface that males have over their female counterparts as shown by greater hand and longer forearm lengths. This creates an increased propelling surface to generate propulsive forces which constitutes a performance advantage in competitive swimming (Troup, 1999; Toussaint, Van Den Berg and Beek, 2002) as propelling efficiency has been shown to increase SL (Kjendlie, Stallman and Stray-Gundersen, 2004).

The males were able to produce greater maximal force production for hand grip and the leg dynamometer ( $p < 0.05$ , Table 2.2) than females. This increased ability to generate force may not fully explain the difference in mechanical power output even though increased force production would be advantageous (Vorontsov, 2011). Increased force production would most likely increase propelling force as well as an increase in power as due to increased capacity and efficiency of the energy delivery system (Sharp, 1986; Vorontsov, 2011). However it seems more important to investigate the time over which the force is applied (Caty et al., 2007) as this provides stronger correlations to swimming velocity and ability (Wirtz et al., 1999).

### *2.5.3 Co-ordination and Stroke Phases*

The most commonly used arm-leg co-ordination pattern in the first lap of the current study was the glide technique with 65 % of swimmers utilising this pattern. The remainder utilised the overlap pattern. The glide co-ordination pattern has previously been reported as characteristic of the 200 m breaststroke event and the overlap technique characteristic of the 50 m breaststroke event (Chollet, Tourny-Chollet and Geizes, 1999; Leblanc, Seifert and Chollet, 2009; Seifert and Chollet, 2009; Soares, Sousa and Vilas-Boas, 1999). In the present study as the participants progressed from the 1<sup>st</sup> to the 4<sup>th</sup> lap, 96 % of the participants altered their arm-leg co-ordination pattern. Of this 96 % who changed, 68 % either moved closer towards the overlap technique or increased the amount of overlap in their technique. The overlap technique is characterised by an overlap of the propulsive phases of the arms and legs it has been suggested that sprint breaststroke swimmers use this technique (Seifert and Chollet, 2005) as it reduces velocity fluctuations which makes the stroke more economical (Vilas-Boas, 1996). These participants could have inverted their co-ordination strategy to move away from the leg kick and placing greater reliance on the arms for propulsion resulting in the reduced glide phase in the majority of participants. Further investigations are required to investigate the shift from the legs to the arms for propulsion during breaststroke swimming. This could be investigated through the analysis of the inter velocity fluctuations of the stroke over each lap of the swim. The remaining participants showed an increase in the amount of glide or a decrease in the overlap in their technique from the 1<sup>st</sup> to 4<sup>th</sup> lap. It could also be postulated that participants altered the timings of the stroke as a result of fatigue. Fatigue has been shown to hamper the sensorimotor system (Forestier and Nougier, 1998; Tripp, Yochem and Timothy, 2007), which affects such functions as awareness, feedback and co-ordination which maintain form and stability. Alterations in these factors may result in the inability to maintain ideal mechanics, thus participants use

compensatory mechanisms of the neuromuscular system to try and maintain homeostasis. However further investigation to substantiate this line of enquiry through the use of electromyography would add greater understanding to neuromuscular responses during a breaststroke swim.

In the present study the inter-lap comparisons show that the participants showed no change in the amount of time spent in the propulsive phases of the stroke (arm pull and leg kick) or the recovery phases of the stroke (arm recovery and leg recovery). Similar results were reported for the co-ordination phases (CPhase 1 and CPhase 2). This may be a failure of using null hypothesis significance testing (NHST) to determining a practical significance of statistical relationships (Cohen, 1992; Cohen, 1994; Osborne, 2008). At a microscopic level the NHST does not consider whether small differences are meaningful.

The findings of the current study cannot be directly related to other breaststroke studies as to the authors knowledge this is the first study that has investigated changes in co-ordination during a 100 m swim in breaststroke. All the previous studies that have investigated the changes in arm-leg co-ordination during a swim have been in front crawl swims (Alberty et al., 2005; Seifert, Chollet and Chatard, 2007; Toussaint et al., 2006). Alberty, et al., (2005) reported an increase in the  $I_{dc}$  in the fatigued conditions which corresponded to a decrease in the non-propulsive phase of the stroke (catch and recovery) with a corresponding increase in the propulsive phases of the stroke (push and pull) this was supported by the findings of Seifert, et al., (2007) who reported greater time spent in the propulsive phase of the stroke in the 4<sup>th</sup> lap compared to the 1<sup>st</sup> lap. It was reported (Toussaint et al., 1988) that this increase in the time spent in the propulsive phases of the stroke may have been as a direct result of a slower hand speed. A slower hand speed has also been linked to a slower swimming velocity. In previous studies an

increase in the time spent in propulsive phases of the stroke was not enough to maintain stroke length and therefore a decrease in swim speed was reported. In the current study this was not the case as there was no increase in the time spent in the propulsive phase of the stroke cycle for either the arms or legs. In breaststroke there is a glide phase for the arms and the legs which may be adequate to allow sufficient recovery, thus maintaining hand speed.

#### *2.5.4 Co-ordination and Stroke Phases Sex Effect*

It is known that female breaststroke swimmers co-ordinate their arms and legs differently to males however this has only been reported in changes of pace from 50 to 200 m breaststroke performance (Seifert and Chollet, 2005). Males have been reported to increase the propulsive phase and reduce the glide phase of the stroke with females arm glide and a smaller propulsive phase (Seifert and Chollet, 2005). In the current study females adopted a motor co-ordination pattern that was characterised with a negative CPhase 1 over all four laps of the swim compared to males that started with a glide co-ordination technique that altered towards the overlap technique from the 1<sup>st</sup> to 4<sup>th</sup> lap. This is different to previous findings of Seifert & Chollet, (2005) who reported that males had significantly shorter glide times compared to females. The differences in findings between that of Seifert & Chollet, (2005) study could be due to the fact that they used national finalist or internationally ranked swimmers compared to elite club swimmers used in the current study. In the current study there was no significant difference between sex in the time spent in any of the phases of the stroke which again is not consistent with Seifert & Chollet, (2005) who reported that males spend significantly ( $p < 0.05$ ) longer in the propulsive phase of the stroke. In these previous studies authors have measured changes in arm-leg co-ordination of the stroke using pre-determined speeds corresponding to 50-100 and 200 m swims. These studies have only investigated performance on a single length of a swim and have

used swim speeds corresponding to 50, 100 or 200 m performance. Compared to the current study where performance was looked at over each lap of the swim. In the current study there was no difference between sexes regards the time spent in the phases of the stroke. This is in contrast to Takagi, et al., (2004) who reported sex differences in simulation propulsion which in the current study was identified with co-ordination phase 1 (CPhase1). The reason for the difference could have been the level of the swimmers used in the study or more likely that in the current study CPhase 1 was investigated over each of the four laps compared to Takagi, et al., (2004) who only investigated co-ordination on the 1<sup>st</sup> lap when the swimmers were not affected by fatigue. This may have been due to the fact that this was a 100 m swim and the significant changes are greater between males and females in the 50 m sprint competitions.

## **2.6 Future Directions**

The current study has given an insight into the co-ordination changes that occur during a 100 m short course swim. The current study is under powered (64 %), ideally a sample size of 46 (90 %) to 50 (95 %) would be used. These numbers are difficult to achieve, due to the small number of breaststroke swimmers that are available. To better understand the reason for the co-ordination changes further research needs to be conducted to investigate the effects of physical fatigue on the swimmers' ability to maintain force and power production throughout the swim. Future studies need to investigate the changes in intra-cyclic velocity fluctuations within and between laps of a swim. This would provide greater understanding of the effects that fatigue has on the swimmers' ability to generate propulsive forces and how changes in co-ordination relate to the swimmers' ability to maintain power output and therefore stroking efficiency.

## **2.7. Conclusion**

This study has investigated changes in stroke kinematics over the duration of a 100 m breaststroke swim in both males and females. Inter lap comparisons showed that there was a significant ( $p < 0.05$ ) decrease in clean swim speed (CSS) over the duration of the swim with similar changes in both sexes. These inter lap changes can be explained by the accumulation of fatigue throughout the swim which reduced the stroke efficiency of both sexes with a significant decrease in inter lap stroke index (SI). The decrease in stroking efficiency can be related to the significant decrease in force production in both the arms and legs and the significant increases in blood lactate. Stroke rate (SR) showed similar decreases over the 1<sup>st</sup> to the 3<sup>rd</sup> lap of the swim which explains the decrease in CSS. Even though there was a significant decrease in CSS there were no significant changes in the time spent in each of the stroke phases. As a result of fatigue the participants were less able to produce muscle force and therefore maintain power output which probably resulted in a decrease in stroke propulsion and an overall decrease in CSS.

## **CHAPTER 3**

### **CHANGES IN INTRA-CYCLIC VELOCITY FLUCTUATIONS AND ARM-LEG CO- ORDINATION DURING A 100 M SHORT COURSE BREASTSTROKE SWIM**



## CHAPTER 3

### Changes in Intra-cyclic Velocity Fluctuations and Arm-Leg Co-ordination during a 100m Short Course Breaststroke Swim

#### 3.1 Abstract

This study investigated changes in kinematics, including intra-cyclic velocity fluctuations and arm-leg co-ordination, over four laps of 100 m short course breaststroke swim from a water start in twelve specialist breaststroke swimmers (males  $n=10$ ) FINA points  $644 \pm 111$ . Intra-cyclic velocity fluctuations were calculated from mean peak velocities of the greater femoral trochanter during the arm and leg propulsive phases and the minimum velocities attained during the Arm-leg lag phase and the recovery of the arms and the legs. Arm-leg co-ordination was defined using two transition phases that described the continuity between the recovery and propulsive phases of the arms and the legs: CPhase 1, corresponding to the time between the end of the leg kick and the start of the arm pull phases; and CPhase 2, corresponding to the time between the end of the arm pull phase and the start of leg recovery phase. The duration of the four stroke phases, (Arm pull, arm recovery, leg kick and leg recovery), the duration of the two transition phases, swim speed, stroke length, stroke rate and velocity fluctuations of the greater femoral trochanter were analysed during the last three strokes of each lap that were not affected by turning or finishing techniques. Each lap was recorded using three 50 Hz standard cameras in underwater housings. Strength indices were assessed using hand grip and isometric leg strength pre and post the swim. A significant decrease in clean swim speed ( $p = 0.046$ ) was observed between laps along with a significant decrease in mean minimum velocity of the greater femoral trochanter during the arm and leg recovery phase of the stroke ( $p = 0.039$ ). There was a significant decrease in the strength indices ( $p < 0.05$ ) from pre to post swim. The results indicate that as participants

progressed through the 100 m swim they became fatigued as there was a decrease in clean swim speed and a decrease in the force generating capacity of the muscles of the arms and the legs. Although there was a significant change in clean swim speed there were no significant changes in co-ordination or timings of the phases of the stroke. Even though there was a significant decrease in the force generating capacity of the arms and legs on the strength indicates these were not related to the peak velocity and/or mean propulsive forces of the arms and legs

### 3.2 Introduction

All the competitive swimming strokes are covered by the FINA rules; for the breaststroke it states that the arms and the legs must have a simultaneous action and the head must appear above the water surface at least once in each stroke cycle (Table 1.1, Section 1.2). The breaststroke swimmer is influenced by relatively large fluctuations in horizontal velocity when compared to front crawl, backstroke and butterfly (Craig and Pendergast, 1979; Holmer, 1979; Maglischo, Maglischo and Santos, 1987). These speed fluctuations are a consequence of the intermittent application of propulsive phases during the stroke cycle (Bober and Czabanski, 1975; Capitao et al., 2006; Craig, Termin and Pendergast, 2006; D'Acquisto and Costill, 1998). The breaststroke stroke has been characterised as having two phases of acceleration, one which is associated with the movements of the arms, the other with the relative movements of the legs (Barbosa et al., 2010; Craig, Termin and Pendergast, 2006; Tourny et al., 1992; Vilas-Boas and Santos, 1994). It has been suggested that there may be a third phase of the stroke that is propulsive whereby some breaststroke swimmers may experience a period of propulsion during the recovery phase of both the arms and the legs (Colman et al., 1998; Maglischo, Maglischo and Santos, 1987; Vilas-Boas, Fernandes and Barbosa, 2010).

A swimmer performs mechanical work predominantly against the force of hydrodynamic resistance (Vorontsov, 2011). The greater the swimmer's velocity the larger the hydrodynamic resistance that the swimmer must overcome, with hydrodynamic resistance increasing proportionally to the square of the swimmers velocity (Toussaint and Truijens, 2005). Therefore, increased swimming velocity requires a relative amplification of the applied muscle force, which increases the propelling force as well as increasing the capacity

and efficiency of the energy delivery system (Vorontsov, 2011). Thus there is a strong rational for a relationship between strength and swimming performance (Sharp, 1986; Vorontsov, 2011). The ability of the swimmer to produce high peak propulsion, high mean propulsion, to reduce drag and to reduce the power lost when giving water kinetic energy is considered the main characteristic with regards success in swimming events (Vilas-Boas, Fernandes and Barbosa, 2010; Toussaint and Truijens, 2005).

Velocity fluctuations occur in the stroke cycle as a result of the change in dominance between propulsive and resistive forces throughout the stroke cycle. The two main phases of the stroke cycle where the swimmer is able to generate propulsive forces that are greater than the resistive forces are during the leg kick and the arm pull phases of the stroke (Colman et al., 1998; Leblanc et al., 2007; Takagi et al., 2004; Vilas-Boas, 1996). During these two phases there will be acceleration of the body's linear velocity. The two phases of the stroke when the resistive forces are greater than the propulsive forces, such that the swimmer would be in a non-propulsive phase (Bober and Czabanski, 1975; Kent and Atha, 1975), are: 1) the time between the end of the leg kick and the start of the out-sweep of the arm stroke (Capitao et al., 2006; D'Acquisto and Costill, 1988) which has previously been termed the arm-leg lag phase (Leblanc et al., 2007); and 2) during recovery when there is forward movement of the arms and legs (Leblanc et al., 2007). During these two phases there will be a deceleration in the body's linear velocity due to the changes in the head, trunk and limb position that hinder the body's motion (D'Acquisto and Costill, 1998). It is important to investigate linear velocity fluctuations to indicate how effective application of propulsive forces are applied by each swimmer as this has been previously been shown to determine swimmers success in competition (Changlur and Brown, 1992; Takagi et al., 2004). The study of linear velocity is well accepted as a relevant area that can enhance swimming performance and its study is

needed to understand the constraints affecting the evolution of swimming performance (Vilas-Boas, Fernandes and Barbosa, 2010).

The study of linear velocity is well accepted as a relevant area that can enhance swimming performance and its study is needed to understand the constraints affecting the evolution of swimming performance (Vilas-Boas, Fernandes and Barbosa, 2010). There are two main methods for quantifying linear velocity; this is either by using a fixed point on the swimmer's trunk, for example the hip joint (Hahn and Krug, 1992; Manley and Atha, 1992), or by using the swimmer's CM (Colman et al., 1998; Figueiredo et al., 2009; Mason, Trong and Richards, 1992; Psycharakis and Sanders, 2009).

The linear velocity of the hip can be assessed in a two ways. The first method is through the use of a mechanical device (Karpovich and Karpovich, 1970; Leblanc et al., 2007; Lima et al., 2006). These mechanical devices are fixed to the pool side at one end the other end is attached directly to the swimmer via a belt, normally in the hip region. This form of assessment only allows the measurement of changes in a swimmer's movements away from the mechanical device. A limitation of the mechanical system is that it does not allow for assessment throughout a swim, as only a single length is monitored. Another method of assessment of tracking a single point on the swimmer is through measurements from video images. This method is not without its limitations, including image distortions due to the aquatic environment, digitisation and calibration errors (Vilas-Boas, Fernandes and Barbosa, 2010). The video imaging system does allow the assessment of linear velocity throughout the duration of a swim. The single point digitisation method does allow multiple strokes to be analysed, which allows for the assessment of inter-stroke variability (Schnitzler et al., 2010) and the assessment of fatigue effects (Alberty et al., 2005).

Assessment of the 3-D movements of the CM has been suggested to be a more accurate method to assess the linear velocity of a swimmer, than fixed point methods (Figueiredo et al., 2009; Psycharakis and Sanders, 2009). Several authors (Figueiredo et al., 2009; Psycharakis and Sanders, 2009) have shown that the intra-cyclic fluctuations of the hip and the centre of mass are different. These authors advocate the use of the 3-D reconstruction method as a more valid method. It has been shown in breaststroke and butterfly swimming that the displacement of the hip and the centre of mass differ as the hip displacement does not account for relative movements of other parts of the body (Barbosa et al., 2003; Maglischo, Maglischo and Santos, 1987). The velocity-time curves of the hip are shifted by 3-5% earlier than the centre of mass, and the hip attains peak velocity values 20% and 5% higher than the CM during the propulsive phases of the arms and legs, respectively, with only a 1% difference in velocity during the arm-leg phase (Leblanc et al., 2007). The coefficients of correlation between the two displacement curves have been found to be significant and to follow similar patterns (Maglischo, Maglischo and Santos, 1987). Using the centre of mass method would reveal some differences in the velocity-time curve, but its calculation is more complex and time consuming (Leblanc et al., 2007). There are still limitations in determining intra-cyclic velocity-time curves with the 3-D methodology as it depends on the accuracy of the anatomical model used to compute inter-limb inertial effects (Schnitzler et al., 2010; Winter, 2005). However determination of intra-cyclic velocity-time curves using a fixed point has been shown to be reliable (Costill, Lee and D'Acquisto, 1987).

The hip joint can be tracked as a single point on the swimmer through measurements from video images. This method is not without its limitations, including image distortions due to the aquatic environment, digitisation and calibration errors (Vilas-Boas, Fernandes and Barbosa, 2010). However the video imaging system does allow the assessment of linear velocity throughout the duration of a swim and allows multiple strokes to be analysed, which

allows for the assessment of inter-stroke variability (Schnitzler et al., 2010) and the assessment of fatigue effects (Alberty et al., 2005) which is not available with the other methods. This method of determination of intra-cyclic velocity-time curves has been shown to be reliable (Costill, Lee and D'Acquisto, 1987).

There are several procedures that have been proposed to quantify the changes in linear velocity throughout a stroke cycle (Leblanc et al., 2007; Psycharakis and Sanders, 2009; Vilas-Boas, 1996). A previous study (Leblanc et al., 2007) which investigated linear velocity changes within a breaststroke stroke cycle reported that linear velocity changes as a ratio of clean mean swim speed.

The previous studies that have investigated changes in hip velocity in breaststroke swimming have investigated changes in intra-cyclic hip velocity over a single length at predetermined race paces corresponding to 50, 100 and 200 m (Leblanc, et al., 2007). Other studies have reported fluctuations in velocity at a strategic place in the swim that is not influenced fatigue (Takagi, et al., 2004). These investigations have not investigated intra lap variations in intra-cyclic velocity, and how this fluctuates and evolves over a race distance. The effect of fatigue on the co-ordination of the arms and legs has previously been reported (Chapter 2), however it is not known how these changes in co-ordination affect the overall propulsion and drag within breaststroke swimming. Investigations into the propulsive forces and resistive forces within the breaststroke have previously been investigated (Persyn et al., 1986; van Tilborgh, Willems & Persyn, 1988; Vilas-Boas, 1994). These authors measured the resultant impulses for the propulsive phase of the leg kick and the arm pull along with the resistive phase of the arm and leg recovery and the glide phase within the stroke cycle. None of the previous studies have investigated how the resistive and propulsive forces change over

the duration of a swim as they have all investigated a section of a swim. The authors have reported that the measurement of the resistive and propulsive forces of a stroke cycle are important as it provides details of the strongest and weakest points of the stroke cycle allowing improvements to be made in the swimmers technique (Barbosa et al., 2002). This process of evaluating the stroke cycle can be a useful diagnostic tool in optimising co-ordination, body positioning and the stroke mechanics of a swimmer (Barbosa et al., 2002).

A study by D'Acquisto & Costill, (1988) concluded that the fastest swimmers were the swimmers that were able to generate the highest peak horizontal velocities during the leg and arm propulsion phases. It has been reported that strength variables should be related to clean swim speeds and not overall performance time as this is a more valid indication of performance (Kjendlie & Stallman, 2011). Previous research (Zampagni et al., 2008) has reported an ability to predict 84% of performance variation using age, height and hand grip strength in 50 m freestyle masters swimmers ( $56 \pm 12$  yrs).. In breaststroke there are two phase of propulsion one from the arms and one from the legs, therefore it is conceivable that that strength measures of both the legs and the arms should be investigated in relation to clean swim speed in breaststroke swimmers. Previous research in breaststroke has reported significant decreases ( $p < 0.05$ ) in isometric hand grip and leg strength (Chapter 2) values from rest to post swimming. However these values were then related to clean swim speed, not to propulsive forces of the arms and legs. Whilst the relationship between a decrease in force generating capacity and fatigue are well known (Toussaint et al., 2006), no previous studies have investigated the relationships between strength measures, IVV and propulsion.



There is conflicting evidence in the literature as to when the peak velocities occur during the breaststroke cycle (Seifert et al., 2010). Some authors have reported greatest peak velocity during the leg propulsion phase (Capitao et al., 2006; Craig, Termin and Pendergast, 2006; D'Acquisto and Costill, 1998), other authors have reported the peak velocity during the arm propulsion phase of the stroke cycle (Maglischo, Maglischo and Santos, 1987; Manley and Atha, 1992) and some have reported similar peak values during the leg and arm propulsive phase of the stroke cycle (Bober and Czabanski, 1975; Kent and Atha, 1975; Tourny et al., 1992). The differences reported in the literature could be due to the techniques used to calculate peak velocity or due to technical characteristics of the swimmers (Maglischo, 2003; Seifert et al., 2010). One of the possible reasons could be that breaststroke swimmers can be either categorised as arm or leg dominant swimmers depending on the phase of the stroke cycle where peak linear velocity is recorded (Maglischo, 2003; Seifert et al., 2010).

If a swimmer maintains a more uniform velocity throughout the stroke cycle, then less energy will be wasted, such that intra-cyclic velocity variation in the stroke can be used to assess the movement efficiency of the swimmer (Kornecki and Bober, 1978). None of the previous research described previously has looked at the changes in IVV throughout a swim. The investigation of these changes in IVV throughout a swim could provide greater insight into the technical changes that are taking place within a swim. These changes could be related to a decrease in force production capacity in the arms and/or legs, depending on the type of breaststroke swimmer, and could help explain the co-ordination changes that have been shown to take place within a 100 m breaststroke swim (Oxford et al., 2010; Takagi et al., 2004).

In breaststroke the relationship between the timing of the propulsive phases of the stroke cycle is an important research area that could influence swimming coaching and performance (Barbosa et al., 2011). Previous research (Chollet, Tourny-Chollet and Geizes, 1999; Chollet et al., 2004; Leblanc et al., 2005; Leblanc, Seifert and Chollet, 2009) has reported a significant relationship between the co-ordination of arms and the legs and linear velocity. It has also been suggested (Tourny et al., 1992) that higher linear velocity might be achieved by reducing the glide phase of the stroke cycle. There is still a need for further research to understand the performance implications of linear velocity (Vilas-Boas, Fernandes and Barbosa, 2010)

To the authors knowledge no previous study has investigated the changes in intra-cyclic velocity fluctuations and corresponding alterations in co-ordination of the arms and legs that have been shown to occur during a 100 m breaststroke swim. By establishing the changes in intra-cyclic velocity fluctuations and co-ordination changes in a swim this would provide the coach and swimmer with a greater understanding of the technical changes that are occurring during a swim and would allow coaches to design suitable interventions to reduce intra-cyclic velocity variations, to help maintain a higher linear velocity throughout a swim, thus enhancing overall performance.

### *3.2.1 Aims*

The aims of this study were to: (1) determine the intra-cyclic velocity fluctuation changes that occur during a 100 m short course breaststroke swim; (2) compare kinematic variables clean swim speed (CSS), stroke rate (SR), stroke length (SL) and stroke index (SI) between each of the four laps as the swimmers progress through the 100 m swim; (3)

establish co-ordination changes during a 100 m short course breaststroke swim; (4) correlate changes in intra-cyclic velocity fluctuations and co-ordination changes over the four laps of the swim; (5) to establish changes in strength following a 100 m short course breaststroke swim ; (6) correlate changes in intra-cyclic velocity from the 1<sup>st</sup> to the 4<sup>th</sup> lap with changes in strength indicators.

### 3.2.2 *Hypotheses*

It was hypothesised that there would be a change in intra-cyclic velocity fluctuations as the swimmer progressed through the swim, and that co-ordination of the arms and legs would change, to reduce the glide phase or increase the overlap, to help maintain clean swim speed as the swim progressed and the swimmer became fatigued.

It was hypothesised that there would be a decrease in strength as shown by decreases in hand grip and leg dynamometry from rest to post 100 m breaststroke swim, and that this decrease would relate to a reduction in propulsive forces in both the arms and legs causing changes in the intra-cyclic velocity.

It was hypothesised that the changes in the co-ordination phases of the stroke would be related to the changes in the propulsive and resistive forces experienced.

### 3.3 Methods

#### 3.3.1 Participants

Ten specialist male breaststroke participants (age  $19.6 \pm 1.3$  years; height  $1.80 \pm 0.05$  m; body mass  $73.6 \pm 6.1$  kg;  $75.24 \pm 7.64$  100m performance time; mean  $\pm$  SD ) volunteered to participate in this study. The selection criterion of the study was that participants had to be competitive at County standard or above within that season within the 100 m breaststroke event. Season best time reported as FINA points  $644 \pm 111$  based on FINA points scoring 2010 for 100 m short course (Splash; FINA point calculator; GeoLogix, Bern, Switzerland). The study was approved by Coventry University's Ethics Committee. The procedures, demands and potential risks of the study were explained to participants and each participant provided written informed consent. Anthropometric measures of height and body mass were made following the International Society for the Advancement of Kinanthropometry (ISAK) protocol (Norton et al., 1996).

Before filming, the skin overlaying the joint centres (lateral malleolus, lateral femoral condyle, greater femoral trochanter, styloid process, epicondyle of humerus and acromion process) were marked on both sides of the body using black PVC electrical tape (19 mm in width) to help identify their location. Joint centres were marked on both sides of the body as the right side of the body was used for qualitative analysis (Quintic, V17; Warwick, UK) on laps 1 and 3 and the left side of the body was analysed on laps 2 and 4. It was assumed that the stroke was symmetrical (Maglischo, 2003). White cloth tape (50 mm in width) cut into 50 mm square sections was used to identify the greater femoral trochanter on the left and right side of the body for 2D analysis (Quintic, V17; Warwick, UK).

### 3.3.2 *Isometric Strength Testing*

#### 3.3.2.1 *Hand Grip*

The maximal handgrip strength of the left and right hand was measured using a grip strength dynamometer (Lafayette 7801, Lafayette, NI, USA). The grip range was adjusted such that the proximal interphalangeal joint of the index finger was flexed at 90 degrees. Measurements were made with the participants standing with the elbow comfortably straight and the wrist in mid-pronation and the arm in full extension above the shoulder. The participants were instructed to maximally grip the dynamometer on each effort, whilst rotating the arm about an anterior-posterior axis to the diametrically opposite position. No involvement of the non-exercising arm was allowed. Before the swim trial three consecutive measures of grip strength were obtained for each hand with a one-minute standing rest interval between measures. This rest period has previously been shown to result in less fatigue (7%) than 15 and 30 seconds rest periods (10 and 12% respectively) (Trossman and Li, 1989). The scale of the dynamometer indicated hand grip strength in kilograms. The maximal grip strength of the three trials was calculated in Newtons by multiplying the dynamometer index by  $9.81\text{m}\cdot\text{s}^{-1}$ . The intraclass correlation coefficient (Thomas and Nelson, 2001) was used to determine the repeatability of the three trials for the left hand grip (97 %) and right hand grip (97 %) at rest.

#### 3.3.2.2 *Leg Strength*

The maximal leg strength was measured using a leg dynamometer (Takei, model T.K.K. 5402, Tokyo, Japan). Unshod participants were required to stand on the platform of the Takei dynamometer with the trunk erect and the knees flexed at an angle of approximately 135 degrees (Coldwells, Atkinson and Reilly, 1994). Knee angle was set by placing a goniometer (Bodycare Southam, England) on the fulcrum (lateral epicondyle of the femur); the stable arm was set

parallel to the lateral midline of the femur and the movement arm was set parallel to the lateral midline of the fibula. The participants reached down with elbows fully extended and gripped the pull bar, then the chain length was adjusted appropriately (Coldwells, Atkinson and Reilly, 1994). The participants were instructed to slowly exert as much force on the handle as possible whilst extending the knees, without using the back muscles, over a period of 10 seconds. The peak force was recorded. Before the swim trial three consecutive measures of leg strength were obtained with a one-minute interval between measures (Trossman and Li, 1989) with the highest score of the three pulls being recorded. The intraclass correlation coefficient (Thomas and Nelson, 2001) was used to determine the repeatability of the three trials for the leg dynamometer (97%) at rest.

Immediately following the 100 m swim trial the participants exited the swimming pool and performed one trial of the isometric leg dynamometer test followed by one trial of hand grip strength test for each hand to give a maximal value. The trials were not repeated 3 times as this would give the swimmer time to recover from the swim and would give improved performance results. Verbal encouragement was given consistently for each trial pre and post swim.

### *3.3.3 Physiological Measurements*

Heart rate was measured following 15 minutes of seated rest (Polar Sports Tester, Polar Electro, Kempele, Finland) and 25µl of capillary blood was taken from an earlobe and analysed using Lactate Pro analyser (Arkray 1710, Kyoto, Japan) in accordance with BASES Guidelines (1997). Heart rate was taken again immediately after the subjects had completed the pre swim isometric strength test and blood lactate was sampled again 5 minutes post the 100 m swim trial.

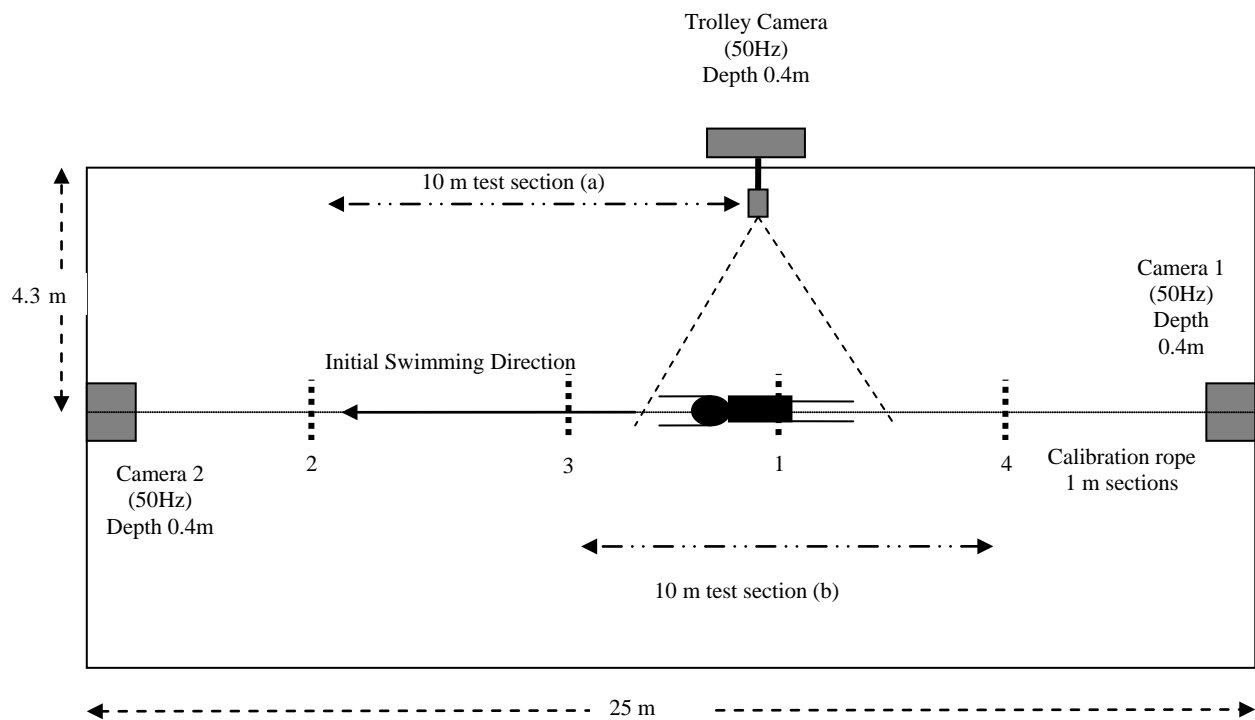
Rating of perceived exertion (RPE) using a scale of 6-20 (Borg, 1998) were recorded immediately the participants left the water after the swim trial.

#### 3.3.4 *Swim Trials*

Each participant performed a self-selected 800 m warm-up in a 25 m pool (Thompson et al., 2003). Each participant was then instructed to perform a maximal 100 m swim trial from a water start with no pre conceived pacing strategies.

Three cameras were used to film the 100 m swim trial. Cameras 1 and 2 (Sony video DCR-TRV460E, Sony, Tokyo, Japan), sampled at 50Hz with the sports preset mode selected (camera preset used in sports mode as shutter speed option not available in camera model), were enclosed in a custom made waterproof housing (Plate 1.0; Chapter 2) at each end of the lane at a depth of 0.4 m, to record frontal and rear views (Figure 3.1). Camera 3 was a waterproof bullet camera, which was suspended underwater from the trolley at a depth of 0.4 m and connected to a visual display unit (VDU) (Sony digital video cassette recorder GV-D800E, Sony, Tokyo, Japan) which was attached to a trolley (Figure 3.1). The field of view was adjusted so that the whole body of the participant was visible (Figure 3.1). The frontal and rear views of the participant were synchronised to the sagittal view (Qunitic, V17, Warwick, UK) using a custom made LED light trigger system that could be seen in all camera views. The trolley was manually moved at the same speed as the participants parallel to the greater femoral trochanter to maintain the joint marker in the approximate centre of the field of view (Figure 3.1), in the sagittal plane, throughout the entire 100 m swim. Refraction due to water was not accounted for in this study as refraction in water due to light rays is relatively low, with previously reported values that vary between 0.39 and 1.28 % (Gourgoulis et al., 2008).

To scale the recorded video footage a calibration rope, with markers located every meter, was suspended horizontally in the water 1 m directly beneath the participants via lane rope suction pads (Figure 3.1). Time to complete 100 m was recorded via a video analysis package (Qunitic, V17, Warwick, UK) as the time from when the feet left the wall at the start until the double hand touch on the wall at the end of the race (to the nearest 0.02s).



**Figure 3.1** Plan view of the two-dimensional filming set-up. 10 m sections, (a) lap1 and 3 (b) laps 2 and 4 were used to determine stroke kinematics.

### 3.3.5 Stroke Parameters

The following stroke parameters were calculated from the 10 m testing sections of the pool on each of the four laps of a 100 m swim from the sagittal view video recordings (trolley camera, Figure 3.1). *Clean Swim speed* ( $\text{m}\cdot\text{s}^{-1}$ ) was defined as the mean forward speed of the

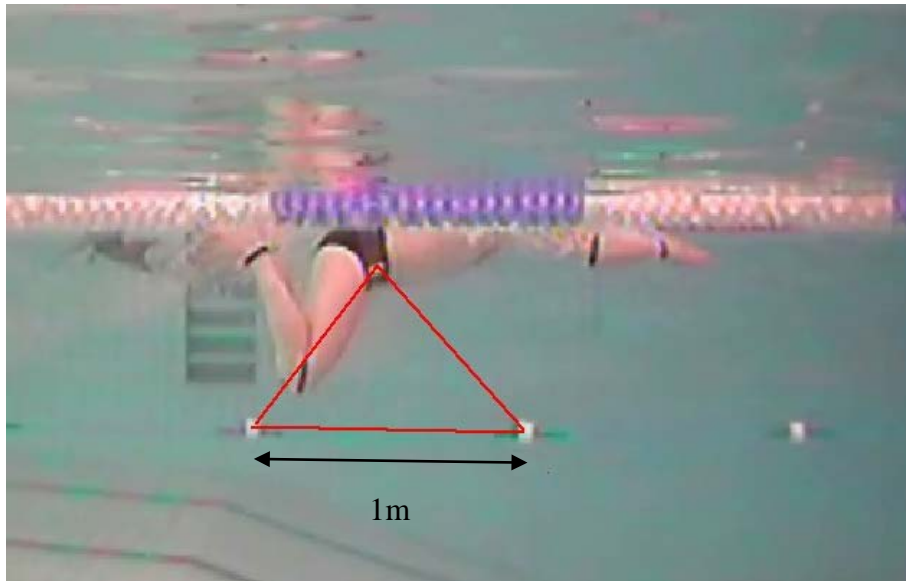


greater trochanter over the 10 m testing section on each lap (Figure 3.1) monitored from time the greater trochanter entered the 10 m testing section until it left the 10 m testing section (Figure 3.1) via the stop clock located on the video analysis software (Qunitic, V17, Warwick, UK) (to the nearest  $0.01 \text{ m}\cdot\text{s}^{-1}$ ); *Stroke frequency* ( $\text{stroke}\cdot\text{min}^{-1}$ ) was defined as the number of stroke cycles performed in one minute, calculated for each 10 m testing section (Figure 2.1) (to the nearest  $0.01 \text{ strokes}\cdot\text{min}^{-1}$ ); *Stroke length* ( $\text{m}\cdot\text{cycle}^{-1}$ ) was defined as the distance that the participants greater trochanter travelled in one stroke cycle, computed from the mean clean swim speed and the mean stroke frequency values (to the nearest 0.01m) (Equation 2.1, Section 2.3.5); *Stroke Cycle Time* (s) was defined as the time taken to complete one complete stroke cycle, calculated as the mean stroke cycle time over each 10 m testing section (to the nearest 0.02 s); Stroke index (SI) as defined by Costill et al., (1985) (Equation 2.2, Section 2.3.5) was used to evaluate swimming efficiency. All stroke parameter calculations were taken from the 10 m testing sections of the pool (Figure 2.1) for each of the four laps of the 100 m swim.

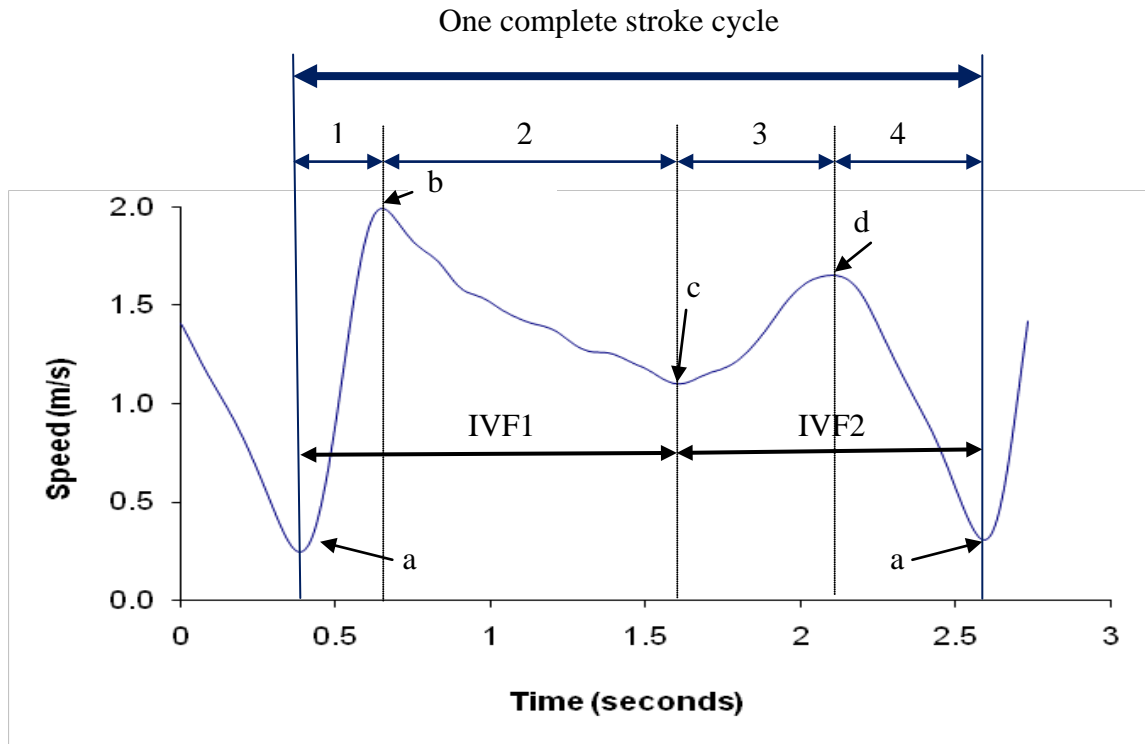
### 3.3.6 Data Processing

The digital video footage was transferred to a laptop computer and analysed using Qunitic Biomechanics system V17 software (Qunitic, V17, Warwick, UK). The last three consecutive strokes (i.e., from the first backwards movement of the feet (the point where the legs were maximally flexed at the start) to the start of the fourth cycle of backward movement of the feet) in the 10 m section of each lap for each participant were selected for analysis. The estimated locations of the greater trochanter along with two markers separated by one meter on the calibration rope were digitised in each frame of video, taken at 50Hz, to obtain the displacement of the greater trochanter in each frame in relation to the calibration rope (Figure 3.2). This was

repeated for each frame to be able to calculate displacement. This method of calculating displacement allowed for changes in speed of the trolley camera in relation to the participants swimming speed. Consequently the time taken by the participant could be calculated by determining the number of frames taken to move a known distance in the pool. From the corresponding displacement data the velocity-time curve was determined for each stroke cycle. The corresponding velocity-time curves were smoothed in an Excel spreadsheet (Hopkins, 2009) via a fourth-order zero-lag Butterworth filter with a frequency cut off of 6 Hz (Takagi et al., 2004; Winter, 2005). Reliability was assessed by calculating the intra-tester correlation of two traces of the same 3 strokes for the hip joint marker and the calibration rope markers for one swimmer ( $R > 0.95$ ). Four velocities were calculated: Peak leg velocity (Leg V; the peak hip velocity recorded during the leg kick; Figure 3.3); Arm-leg lag velocity (Lag V; the minimum hip velocity recorded during the phase between the start of leg propulsion and the start of the arm propulsion phase; Figure 3.3); Arm velocity (Arm V; the peak hip velocity recorded during the arm propulsion phase; Figure 3.1) and Recovery V (Rec V; the minimum hip velocity recorded during the arm and leg recovery phase; Figure 3.1). From these hip velocities intra-velocity fluctuations of the stroke cycle were calculated (IVF1; Equation 3.3, Figure 3.1 and IVF 2; Equation 3.2, Figure 3.3). Intra-cyclic velocity fluctuation (IVF) was calculated (Leblanc, et al., 2007; Vilas-Boas, 1996; Equation 3.3) and expressed as a percentage of mean velocity for the three stroke cycles.



**Figure 3.2** Calibration of displacement of the greater femoral trochanter. How the displacement of the hip marker was determined from the calibration rope.



**Figure 3.3** Represents an illustration of a typical time velocity curve for breaststroke swimming. A complete breaststroke stroke cycle with indication of where in the stroke cycle velocities were calculated (a = minimum hip velocity during arm and leg recovery (Rec V) ; b = peak hip velocity during the leg kick (Leg V); c = minimum hip velocity following the Leg V and before Arm V (Lag V); d = peak hip velocity during the arm pull (Arm V); 1 = phase 1 of the stroke cycle corresponding to the leg push; 2 = phase 2 which is the time between the end of the leg push and the start of the arm pull phase; 3 = phase 3 which, corresponds to the arm pull phase; and 4 = phase 4 of the stroke cycle which corresponds to the arm push and leg pull phases.

Equation 3.1

$$\text{IVF1} = \text{Leg V} - \text{ALL V}$$

Equation 3.2

$$\mathbf{IVF2 = Arm\ V - Rec\ V}$$

Equation 3.3

$$\mathbf{IVF\ (\%) = \frac{Mean\ V}{(Leg\ V - ALL\ V) + (Arm\ V - Rec\ V)}}$$

The acceleration (Equation 3.3) of the greater trochanter was calculated for each of the four phases (Figure 3.1) using the minimum and maximum velocities recorded during each of the four phases (Figure 3.1). The acceleration values obtained (Equation 3.4) for each of the four phases (Figure 3.1) and the mass (kg) of the participant were used to calculate (Equation 3.5) the mean net propulsive force during phase 1 and phase 3 and net mean resistive forces (Equation 3.5) during phase 2 and phase 4 (Figure 3.1) for each of the 4 laps.

Equation 3.4

$$\mathbf{a = \frac{(V_f - V_i)}{t}}$$

Equation 3.4

$$\mathbf{F = m. a}$$

### 3.3.7 *Arm and Leg Co-ordination and Stroke Phases*

Three complete stroke cycles (Chollet et al., 2004), which were completed within the 10 m testing section of each of the four laps prior to the turn or the finish, were analysed using the synchronised frontal and sagittal camera views (Figure 3.1) to determine the average duration of each of the following phases: Arm Pull (the time between the separation of the hands from the extended position in front of the body until the first forward movement of the elbow when the hands were under the head); Arm recovery (the time between the end of the arm pull phase and the start of the separation of the hands from the extended position); Leg kick (the time between the start of the first backwards movement of the feet, the point where the legs were maximally flexed at the start, and the point when the legs were fully extended); Leg recovery (the time between the end of the leg kick phase and the complete flexion of the knee until forward movement of the feet had finished); Co-ordination phase 1 (CPhase1) was calculated as the time between the end of the leg kick phase and the start of the arm recovery phase and was used to classify the participants co-ordination as overlap (represented by a negative value to the nearest 0.02 s indicating simultaneous propulsion of the arms and legs), glide (represented by a positive value to the nearest 0.02 s indicating a delay (glide) in the initiation of the arm pull phase) or continuous; Co-ordination phase 2 (CPhase 2; the time between the end of the arm pull phase and the start of leg kick phase); Arm lag time (ALT; corresponded to the time from the start of the leg kick to the beginning of arm pull). All phases were expressed as a percentage of total cycle time with a precision of 0.02 s (Figure 2.2). It should be noted that the start of the arm pull phase and the end of the arm pull phase as described above does not necessarily correspond the start and end of the propulsive components of the arms' stroke, similarly for the leg kick, the start of the leg kick and the end of the leg kick does not necessarily correspond to the start and end of the leg propulsion (Maglischo, 2003). The key stroke phases of the arms and legs were subjectively

determined by three independent operators using a blind technique i.e. without knowing the results of the other operators. The three independent analyses were then compared with the mean difference of the operators being ( $< 0.04$  to the nearest  $0.02$  s), which was less than the  $0.04$  s which has previously been used to validate key stroke phases (Seifert, Chollet and Chatard, 2007).

### 3.3.8 *Statistical Procedures*

Statistical analyses were conducted using SPSS version 16.0 (SPSS, Inc., Chicago, IL, USA). Standard statistical methods were used to calculate mean and standard deviation for all measured variables. Normal distribution of the data was verified using Levenes's test. The percentage data was tested for normal distribution using the z-score of skewness and kurtosis in accordance with Vincent, (2005). If normal distribution was not reported then data was corrected for heterogeneity of variance using the Arcsine transformation (Black, 1999). The level of significance was set at  $p < 0.05$ . One-way Analysis of variance with HSD Tukey post-hoc tests were used with lap (1, 2, 3 and 4) as the factors to compare selected variables at the same point of each of the four laps. The effect size was estimated using partial Eta squared ( $\eta p^2$ ) for analysis of variance. Since this method is likely to overestimate effect sizes, values were interpreted according to Ferguson (2009) as no effect if  $0 \leq \eta p^2 < 0.05$ ; a minimum effect if  $0.05 \leq \eta p^2 < 0.26$ ; a moderate effect if  $0.26 \leq \eta p^2 < 0.64$ ; and a strong effect if  $\eta p^2 \geq 0.64$ . Paired T tests were used to compare pre and post for hand grip strength, isometric leg strength and other physiological tests. The effect size of the paired T test was estimated using Pearson's correlation coefficient in accordance with (Rosnow and Rosenthal, 2005), values were interpreted according to Cohen, (1988) as  $r = 0.10$  (small effect),  $r = 0.30$  (medium effect) and  $r = 0.50$  (large effect) Effect size results of the paired t tests were

reported in accordance with Field (2009). Pearson product moment correlation coefficients were determined among selected kinematic variables for each of the four laps.

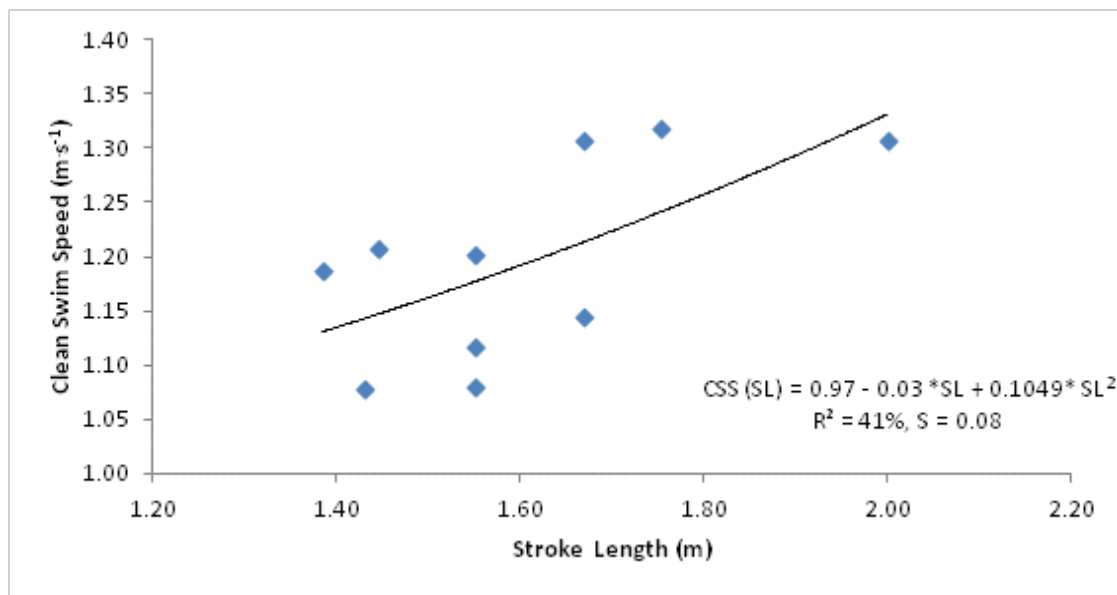
## **3.4 Results**

### *3.4.1 Performance Data*

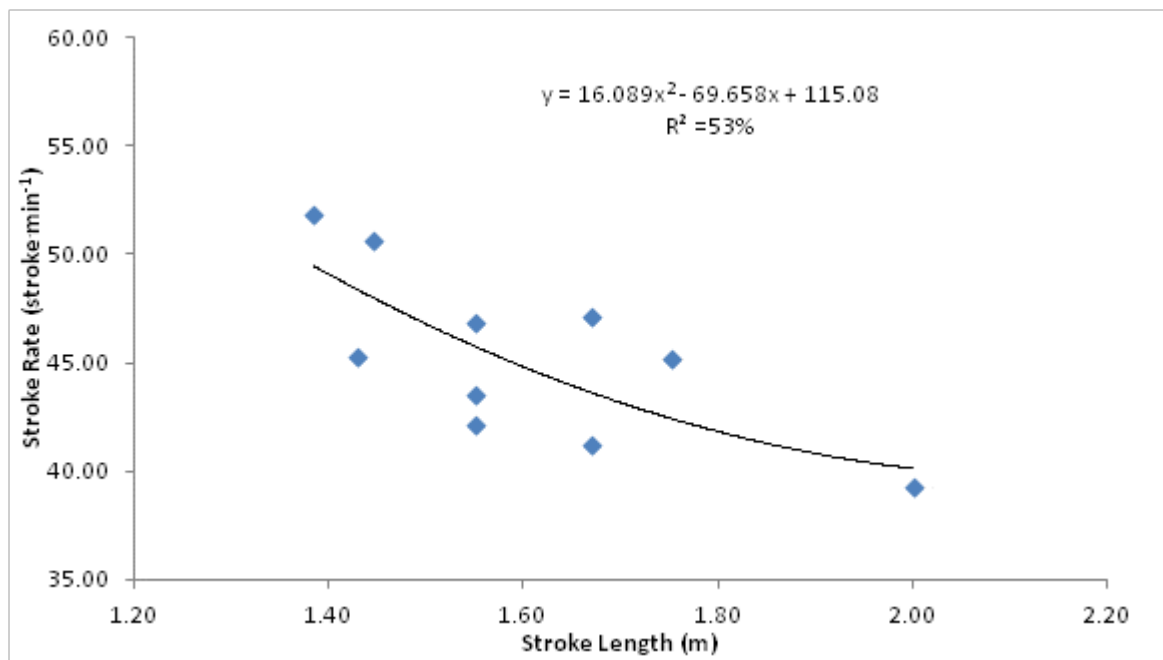
The mean 100 m performance time for the participants (Table 3.1) showed a statistically significant negative correlation ( $r = -.847, p = 0.002$ ) with mean clean swim speed (CSS) achieved across the 100 m swim.

Mean stroke length (SL) across the 100 m swim showed a statistically significant positive correlation with mean CSS ( $r = .642, p = .045$ ) (Figure 3.4) and a statistically significant negative correlation with stroke rate (SR) ( $r = -.711, p = .021$ ) (Figure 3.5)





**Figure 3.4** Relationship between clean swim speed and stroke length over the four laps (n = 10 participants; each data point represents the mean clean swim speed, for a participant, plotted against their mean stroke length over the four laps). The line has been fitted via a quadratic regression analysis.



**Figure 3.5** Relationship between mean stroke length and mean stroke rate over the four laps (n=10 participants; each data point represents the mean stroke rate, for a participant, plotted against their mean stroke length over the four laps). The line has been fitted via a quadratic regression analysis

There was no significant correlation between the mean values for clean swim speed and any of the strength test values ( $p < 0.05$ ). There was no statistically significant relationship between the percentage change in strength values and the percentage change in CSS from the 1<sup>st</sup> to the 4<sup>th</sup> lap.

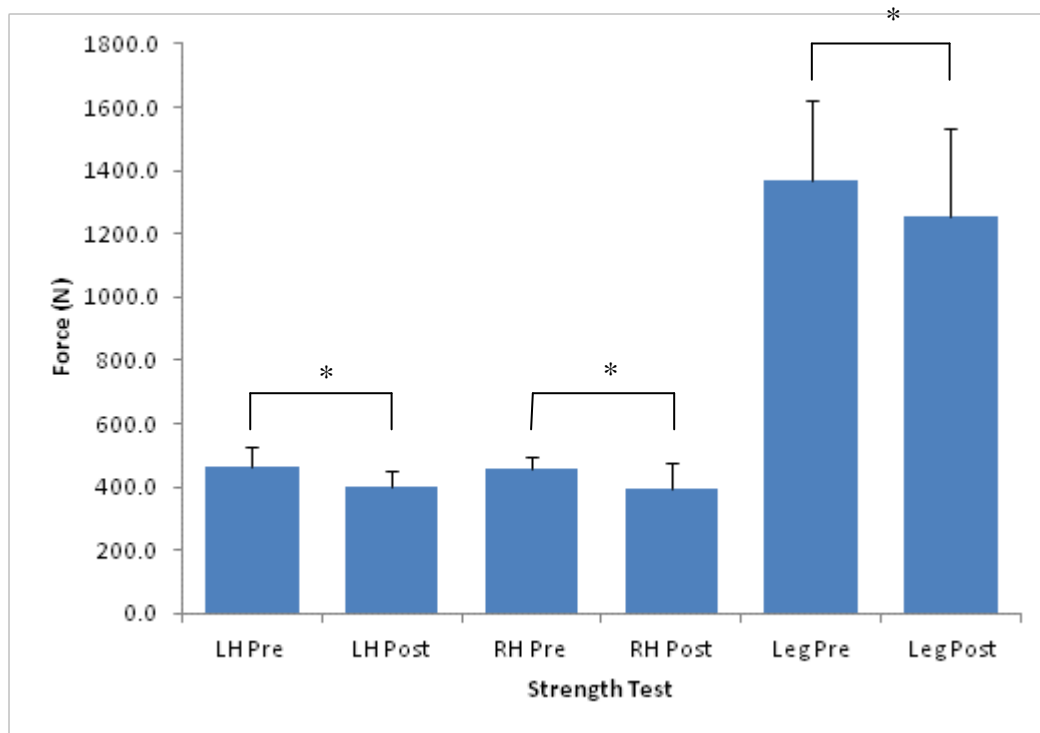
### 3.4.2 Physiological Measures

**Table 3.1** Physiological measures at rest and post 100 m swim of the participants n=10. All values are given as mean  $\pm$  SD.

Resting	Post Swim	Resting	Post Swim	Post Swim
Heart Rate	Heart Rate	Blood lactate	Blood Lactate	RPE
(Beats $\cdot$ min $^{-1}$ )	(Beats $\cdot$ min $^{-1}$ )	(mmol $\cdot$ L $^{-1}$ )	(mmol $\cdot$ L $^{-1}$ )	
73 $\pm$ 14*	185 $\pm$ 13	1.6 $\pm$ 0.3*	8.8 $\pm$ 2.8	18 $\pm$ 1

\*Denotes a statistically significant difference from rest to post swim  $p < 0.05$ .

The physiological measures of heart rate and blood lactate showed significant increases ( $p < 0.05$ , Table 3.1) of 146 % and 450 % respectively from rest to post swim. Hand grip strength of the left hand significantly decreased by  $12.4 \pm 8$  %,  $t$  (9) 3.60,  $p = .006$ ,  $r = 0.77$ , (Figure 3.6) from pre to post swim with a similar significant decrease of  $14.1 \pm 12$  %  $t$  (9) 4.46,  $p = .002$ ,  $r = 0.83$ , (Figure 3.6) for the right hand. Leg strength values also significantly decreased by  $8.6 \pm 5.9$  %,  $t$  (9) 4.61,  $p = .001$ ,  $r = 0.84$ , (Figure 3.6) from pre to post the 100 m swim.



\* Denotes a statistically significant difference in pre and post strength values  $p < 0.05$

**Figure 3.6** Depicts the change in mean  $\pm$  SD strength values for isometric hand grip of the left hand (LH), right hand (RH) and for isometric leg strength pre and post a 100 m breaststroke swim.

### 3.4.3 Stroke Parameters

There was a statistically significant difference in clean swim speed between laps  $F(3,36) = 2.94, p = .046, \eta^2 0.2$ , (Table 3.2). A Tukey post-hoc test failed to reveal significant findings between the laps. There was no statistically significant difference in stroke length (SL)  $F(3,36) = 0.23, p = .874, \eta^2 0.02$  or stroke rate (SR)  $F(3,36) = 0.75, p = .532, \eta^2 0.06$ , between laps (Table 3.2). There was no statistically significant in stroke index (SI) between laps  $F(3,36) = 1.03, p = .393, \eta^2 0.08$  (Table 3.2).

**Table 3.2** Mean(  $\pm$  SD) values and coefficient of variation percentage (CV % ) for Clean swim speed (CSS), stroke length (SL), stroke rate (SR), stroke index (SI) and stroke cycle time for each lap of the 100 m swim.

	<b>1st Lap</b>	<b>2nd Lap</b>	<b>3rd Lap</b>	<b>4th Lap</b>
CSS (m·s <sup>-1</sup> )	1.26 $\pm$ 0.12 (cv=9)	1.22 $\pm$ 0.08 (cv=7)	1.15 $\pm$ 0.09 (cv=8)	1.16 $\pm$ 0.09 (cv=8)
SL (m·cycle <sup>-1</sup> )	1.62 $\pm$ 0.25 (cv=15)	1.63 $\pm$ 0.17 (cv=11)	1.58 $\pm$ 0.19 (cv=12)	1.57 $\pm$ 0.21 (cv=14)
SR(stroke·min <sup>-1</sup> )	47.1 $\pm$ 6.2 (cv=13)	45.3 $\pm$ 4.4 (cv=10)	43.9 $\pm$ 4.1 (cv=9)	44.8 $\pm$ 5.0 (cv=11)
Stroke Cycle time (s)	1.43 $\pm$ 0.14 (cv=10)	1.50 $\pm$ 0.18 (cv=12)	1.53 $\pm$ 0.17 (cv=11)	1.48 $\pm$ 0.16 (cv=11)
Stroke Index	2.06 $\pm$ 0.47 (cv=23)	1.99 $\pm$ 0.30 (cv=15)	1.83 $\pm$ 0.33 (cv=18)	1.82 $\pm$ 0.36 (cv=20)

#### 3.4.4 *Intra-cyclic Hip Velocity Fluctuations*

There was a statistically significant difference in the minimum hip velocity during the recovery phase (phase 4, Figure 3.1) of the stroke cycle between lap  $F(3,36) = .195$ ,  $p = .035$ ,  $\eta^2 = 0.22$  (Table 3.3). A Tukey post-hoc test revealed that phase 4 approached statistical significance when the 1<sup>st</sup> and the 4<sup>th</sup> lap were compared (Tukey  $p = .055$ ). There were no other statistical differences in intra-cyclic hip velocity fluctuations between laps ( $p > 0.05$ ), however there were high levels of variability within the measures (Table 3.3).

**Table 3.3** Mean (  $\pm$  SD) and coefficient of variation percentage (CV %) values of the peak and minimum velocities of the hip during the phases of the stroke.

	<b>1st Lap</b>	<b>2nd Lap</b>	<b>3rd Lap</b>	<b>4th Lap</b>
Kick peak velocity (m's <sup>-1</sup> )	2.11 $\pm$ 0.21 (cv=10)	2.01 $\pm$ 0.21 (cv=11)	1.76 $\pm$ 0.27 (cv=16)	1.88 $\pm$ 0.14 (cv=7)
Arm-Leg Lag Minimum Velocity (m's <sup>-1</sup> )	1.20 $\pm$ 0.30 (cv=25)	1.20 $\pm$ 0.15(cv=12)	1.04 $\pm$ 0.18 (cv=17)	1. 07 $\pm$ 0.23 (cv=22)
Arm peak velocity (m's <sup>-1</sup> )	1.72 $\pm$ 0.22 (cv=13)	1.66 $\pm$ 0.28 (cv=17)	1.53 $\pm$ 0.17 (cv=11)	1.58 $\pm$ 0.19 (cv=12)
Recovery minimum velocity (m's <sup>-1</sup> )	0.23 $\pm$ 0.12* (cv=52)	0.22 $\pm$ 0.10 (cv=46)	0.21 $\pm$ 0.13 (cv=62)	0.11 $\pm$ 0.14* (cv=127)

\*Denotes a statistically significant difference between the 1<sup>st</sup> and 4<sup>th</sup> lap for minimum velocity during the recovery phase  $p < 0.05$

**Table 3.4** Mean ( $\pm$  SD) and coefficient of variation percentage (CV %) values of intra-cyclic velocity fluctuations between peak and minimum velocities of the hip measured in the four phases of the stroke, across the four laps of the 100 m swim. Where: IVF1 represents the intra-cyclic hip velocity fluctuations from phase 1 to phase 2 of the stroke cycle; IVF2 represents the intra-cyclic hip velocity fluctuation between phase 3 and phase 4 of the stroke cycle See Equations 3.0, 3.1 and 3.2 (Section 3.3.3 of the methods).

	<b>1st Lap</b>	<b>2nd Lap</b>	<b>3rd Lap</b>	<b>4th Lap</b>
IVF1 (m·s <sup>-1</sup> )	0.88 $\pm$ 0.25 (cv=28)	0.77 $\pm$ 0.25(cv=33)	0.73 $\pm$ 0.23(cv=32)	0.81 $\pm$ 0.17 (cv=21)
IVF2 (m·s <sup>-1</sup> )	1.52 $\pm$ 0.26 (cv=17)	1.47 $\pm$ 0.26(cv=18)	1.32 $\pm$ 0.24(cv=18)	1.47 $\pm$ 0.22 (cv=15)
IVF %	53 $\pm$ 10 (cv=19)	55 $\pm$ 9 (cv=16)	59 $\pm$ 2 (cv=3)	52 $\pm$ 10 (cv=19)

There was no statistically significant difference in IVF1(intra-cyclic hip velocity fluctuation from phase 1 to phase 2 of the stroke cycle; Figure 3.2, Chapter 3, Section 3, Table 3.4) or IVF2 (intra-cyclic hip velocity fluctuation between phase 3 and phase 4 of the stroke cycle; Figure 3.2) with lap ( $p > 0.05$ ). There was no statistically significant difference in intra-velocity fluctuations relative to CSS (IVF %) between laps  $F(3,44) = 0.10$ ,  $p = .511$ ,  $\eta^2 0.05$ , however there were high levels of variability within the measures (Table 3.4).



**Table 3.5** Mean ( $\pm$  SD) and coefficient of variation percentage (CV %) values of mean resistance and mean propulsion during the phases of the stroke across the four laps of the 100 m swim.

	<b>1st Lap</b>	<b>2nd Lap</b>	<b>3rd Lap</b>	<b>4th Lap</b>
Propulsion Legs (N)	529 $\pm$ 149 (cv=28)	492 $\pm$ 163 (cv=33)	448 $\pm$ 125 (cv=28)	467 $\pm$ 119 (cv=25)
Propulsion Arms (N)	126 $\pm$ 71 (cv=57)	120 $\pm$ 80 (cv=67)	123 $\pm$ 53 (cv=43)	106 $\pm$ 41 (cv=39)
Total Propulsion (N)	655 $\pm$ 191 (cv=29)	612 $\pm$ 215 (cv=35)	570 $\pm$ 167 (cv=29)	573 $\pm$ 145 (cv=25)
Resistance Arm-leg lag (N)	-169 $\pm$ 62 (cv=36)	-155 $\pm$ 70 (cv=45)	-150 $\pm$ 78 (cv=52)	-178 $\pm$ 94 (cv=53)
Resistance Recovery (N)	-345 $\pm$ 128 (cv=37)	-363 $\pm$ 174 (cv=48)	-271 $\pm$ 88 (cv=32)	-338 $\pm$ 116 (cv=34)
Total Resistance (N)	-514 $\pm$ 131 (cv=25)	-518 $\pm$ 219 (cv=42)	-422 $\pm$ 130 (cv=31)	-515 $\pm$ 124 (cv=24)

There was no statistically significant change in mean propulsion of the arms or legs with lap ( $p > 0.05$ , Figure 3.1) this is due to high inter-individual variability. There was no statistically significant change in total propulsion over the duration of the swim. There were no significant ( $p > 0.05$ ) relationships between the propulsion forces of the arms and legs and strength values (Table 3.5).

#### *3.4.5 Co-ordination:*

Three participants utilised the overlap co-ordination technique throughout the 100 m swim, five participants utilised the glide co-ordination technique throughout the 100 m swim and two participants started with the glide co-ordination technique but changed to the overlap co-ordination from the 1<sup>st</sup> to the 4<sup>th</sup> lap.

The durations of the arms and legs phases (Table 3.6) for the whole group showed no significant ( $p > 0.05$ ) change with lap. There were no significant ( $p > 0.05$ ) changes in the co-ordination phases (CPhase 1 and CPhase 2, Table 3.6) over the duration of the swim.

**Table 3.6** Mean ( $\pm$  SD) and coefficient of variation percentage (CV %) for the durations of arm and leg stroke phases and arm-leg co-ordination over the four laps of the 100 m swim.

	<b>1st Lap</b>	<b>2nd Lap</b>	<b>3rd Lap</b>	<b>4th Lap</b>
Leg Kick (s)	0.36 $\pm$ 0.07 (cv=20)	0.36 $\pm$ 0.09 (cv=26)	0.36 $\pm$ 0.08 (cv=23)	0.38 $\pm$ 0.12 (cv=30)
Kick Recovery (s)	1.08 $\pm$ 0.17 (cv=16)	1.14 $\pm$ 0.24 (cv=21)	1.17 $\pm$ 0.23 (cv=20)	1.10 $\pm$ 0.24 (cv=22)
Arm Pull (s)	0.66 $\pm$ 0.10 (cv=15)	0.68 $\pm$ 0.09 (cv=13)	0.70 $\pm$ 0.09 (cv=12)	0.71 $\pm$ 0.07 (cv=10)
Arm Recovery (s)	0.76 $\pm$ 0.17 (cv=22)	0.83 $\pm$ 0.22 (cv=27)	0.82 $\pm$ 0.21 (cv=25)	0.78 $\pm$ 0.21 (cv=26)
Co-ordination Phase 1 (s)	0.05 $\pm$ 0.22 (cv=440)	0.08 $\pm$ 0.28 (cv=350)	0.07 $\pm$ 0.26 (cv=371)	0.01 $\pm$ 0.29 (cv=2900)
Co-ordination Phase 2 (s)	0.37 $\pm$ 0.09 (cv=24)	0.38 $\pm$ 0.08 (cv=21)	0.40 $\pm$ 0.11 (cv=28)	0.38 $\pm$ 0.07 (cv=18)

**Table 3.7** Mean ( $\pm$ SD) and coefficient of variation percentage (CV %) values of arm and leg stroke phases and arm-leg co-ordination expressed as a percentage over the four laps of the 100 m swim.

	1st Lap	2nd Lap	3rd Lap	4th Lap
Leg Kick (%)	25.1 $\pm$ 5.7 (cv=23)	24.8 $\pm$ 8.7 (cv=35)	24.3 $\pm$ 7.4 (cv=30)	26.5 $\pm$ 9.8(cv=37)
Kick Recovery (%)	75.7 $\pm$ 5.2 (cv=7)	75.5 $\pm$ 8.5 (cv=11)	76.2 $\pm$ 7.7 (cv=10)	75.0 $\pm$ 9.8 (cv=13)
Arm Pull (%)	46.8 $\pm$ 8.7 (cv=19)	45.8 $\pm$ 9.7 (cv=21)	46.6 $\pm$ 9.2 (cv=20)	46.9 $\pm$ 8.8 (cv=19)
Arm Recovery (%)	53.2 $\pm$ 8.7 (cv=16)	54.2 $\pm$ 9.7 (cv=18)	53.4 $\pm$ 9.2 (cv=17)	51.7 $\pm$ 9.4 (cv=18)
Co-ordination Phase 1(%)	2.7 $\pm$ 16.1 (cv=596)	3.9 $\pm$ 18.7 (cv=479)	3.0 $\pm$ 17.7 (cv=590)	-0.7 $\pm$ 20.4 (cv=4131)
Co-ordination Phase 2 (%)	25.6 $\pm$ 6.0 (cv=23)	25.5 $\pm$ 4.9 (cv=19)	26.1 $\pm$ 6.5 (cv=25)	26.1 $\pm$ 4.9 (cv=19)
ALT (%)	29.1 $\pm$ 13.0 (cv=45)	27.6 $\pm$ 10.3 (cv=37)	27.2 $\pm$ 9.8 (cv=36)	24.4 $\pm$ 10.4 (cv=42)

There were no statistically significant correlation ( $p > 0.05$ ) between the percentage changes between the 1<sup>st</sup> and the 4<sup>th</sup> lap for any of the co-ordination and intra-velocity variables (Table 3.7), however there were high levels of inter individual variability in the measures.

### 3.5 Discussion

The breaststroke is characterised by large fluctuations in horizontal velocity due to the intermittent application of propulsive phases during the stroke cycle (Bober and Czabanski, 1975; Capitao et al., 2006; Craig, Termin and Pendergast, 2006; Tourny et al., 1992). The results of the current study show that the participants experienced two phases of increased velocity which

coincided with the leg and arm propulsive phases of the stroke cycle (Table 3.4), consistent with previous studies (Bober and Czabanski, 1975; Capitao et al., 2006; Craig, Termin and Pendergast, 2006; Tourny et al., 1992). The overall data suggests the participants were leg dominant as the peak velocity within the stroke cycle occurred during the leg propulsion phase (Table 3.4), in line with previous research (Maglischo, Maglischo and Santos, 1987; Manley and Atha, 1992) however there is intra-individual variation (Table 3.4). The mean peak velocity over the four laps of the swim for the leg kick did not correlate ( $r = 0.03$   $p = 0.928$ ) with CSS which is inconsistent with the previously reported high correlation  $r = 0.81$  (D'Acquisto and Costill, 1998). The difference in the correlation values between the present and previous study could be due to the methods implemented. In the D'Acquisto & Costill, (1998) study they only took the CSS value from the faster of two all-out 20 yd (22.86 m) trials due to the use of a velocity meter being used. In the current study the CSS was the average over the four laps of a 100 m swim. Within the current study there was a significant ( $p = .046$ ) decrease over the four laps indicating that the overall average would be lower than that used in previous studies. In the current study velocity was calculated from a single point analysis of the greater femoral trochanter using 2D video analysis compared to previous studies that used a velocity meter.

The lowest mean minimum velocity over the four laps of the swim was recorded during the arm and leg recovery phase of the stroke cycle which is consistent with previous research (Bober and Czabanski, 1975; Craig, Termin and Pendergast, 2006; Leblanc et al., 2007; Manley and Atha, 1992). During the arm and leg recovery phases of the stroke the participants would be pushing the arms and legs forwards, underwater, increasing the drag forces and causing the greatest loss swim speed. According to Sanders, (1996), during the recovery phase of the legs the thighs reach their

maximal angle of between 54 and 68 degrees in relation to knee joint. This maximum knee angle is just prior to the start of the leg kick phase which puts the swimmers into a position that greatly increases the amount of hydrodynamic resistance, as a result of an increased projected area and therefore increased drag forces, resulting in significantly reduced velocity (Maglischo, 2003). The mean minimum velocity over the 4 laps for the arm-leg lag phase of the stroke cycle (Table 3.3) showed statistically significant correlations ( $r = 0.62$ ,  $p < 0.05$ ) with mean CSS, consistent with previous studies (D'Acquisto and Costill, 1998) who reported correlations of  $r = 0.80$  for minimum velocity recorded prior to the arm propulsion phase.

### *3.5.1 Changes in Kinematics between Laps across the 100 m Swim*

Previous research (Barbosa et al., 2010; Capita et al., 2006; Colman et al., 1998; Leblanc et al., 2007; Vilas-Boas and Santos, 1994) that has looked at intra-velocity fluctuations have only looked at changes over a single 25 m, which does not provide the coach, swimmer or sports scientist with an insight into the affects of the accumulation of fatigue and the subsequent changes that are taking place over a race length swim (Alberty et al., 2005; Pelayo et al., 2007). In the current study the changes in intra-cyclic velocity fluctuations were investigated over the 4 laps of a 100 m swim. The current study has looked to quantify the changes that occurred during a swim as this is an important factor in better understanding overall swimming performance. By investigating these parameters during a swim we can gain a greater depth of understanding into the processes that occur during a swim to better understand why performance (CSS) drops off as swimmers progress through the swimming race. It is not known if these are direct results of the fatigue process or due to technical changes.

The results of the current study show that there are two intra-cyclic velocity fluctuations (IVF) within the stroke cycle (Table 3.4). The first is between phase 1 and phase 2 of the stroke cycle (IVF1, Figure 3.3) and the second is between phases 3 and 4 (IVF2, Figure 3.3) of the stroke cycle. The current findings show that there are no significant ( $p > 0.05$ ) changes in IVF1 or IVF2 as there is no significant ( $p > 0.05$ ) change, across the time course of the swim trial, in the peak velocities that were achieved during the leg kick and the arm pull phases of the stroke cycle.

The findings of the current study show that when intra-velocity fluctuations (IVF) are calculated as a percentage (IVF%) of mean clean swim speed (CSS) (Equation 3.2), as in previous studies (Leblanc et al., 2007; Vilas-Boas, 1996), there is no statistically significant change ( $p > 0.05$ , Table 3.5) over the 4 laps. However, the results of the current study show that as the participants progressed through the swim there was a significant change in the CSS (Table 3.2) over the 4 laps, which is consistent with previous research (Oxford, et al., 2010; Takagi, et al., 2004; Thompson, et al., 2000, Chapter 2, Section 4). As there is a reduction in CSS there must be a decrease in the production of propulsive forces or an increase in the resistive forces, or both, as a swimmer's performance is limited by the effective application of propulsive forces or hydrodynamic resistance (Barbosa et al., 2011; Toussaint and Truijens, 2005). The significant decrease in the strength tests from rest to post swim (Figure 3.6) indicates a loss of force generating capacity. This is further supported with the significant increase in BLa values (Table 3.1). The high values of BLa post swim is a confirmation of the high level of effort that the participants put into the swim and supports glycolytic participation (Figueiredo et al., 2012). High levels of BLa have been attributed to intracellular acidosis which has been shown to be related to decreased glycolytic activity (Danforth, 1965; Mader, Heck and Hollmann, 1983; Trividi and Danforth, 1966). Reduced glycolytic capacity

will decrease the mechanical power of the affected muscles (Fitts, 2003). As the participants are less able to generate force which there will likely be a decrease in the propelling performance, which may be the cause for the changes swimmers, made with regards to the combination of SR and SL. The results suggest that hydrodynamic drag is not increasing as there is no alteration in the rate of change in the participant's linear velocity as the resistive forces have not significantly changed over the duration of the swim, suggesting that overall the participants were able to maintain their stroke timing throughout the swim. This was not expected and we can reject the hypothesis that changes in co-ordination of the arms and legs would occur as the swimmer attempted to maintain swim speed as the swim progressed and the swimmer became fatigued. No previous studies in breaststroke have compared the changes in intra cyclic hip velocity over the duration of a swim. This is an important area of swimming biomechanics, which warrants further investigation with a larger sample and with a better quality of swimmer. Such future research would provide greater insight into what is happening during a swim to better understand why there are decreases in the swimmers' clean swim speed over the duration of a 100 m breaststroke swim (Thompson, Haljand and MacLaren, 2000; Oxford et al., 2010)

The study of the propulsive and resistance forces can provide useful information about the technical proficiency of the swimmer (Alves, Gomes-Pereira and Pereira, 1996). Previous studies have reported that average resultant impulses (Persyn, Colman and Tilborgh, 1992; Van Tilborgh, Eustache and Persyn, 1988; Vilas-Boas and Santos, 1994) can be calculated per stroke and used to identify mechanical inefficiencies within a breaststroke stroke cycle. The results for the current study show that there was no significant change in the participants' propulsive forces over the duration of the swim (Table 3.5).



The findings of the current study show that if the net propulsive forces are broken down into their individual components, which relate to the individual phases (phases 1 and 3), then the majority of the net propulsive force comes from the legs rather than the arms (Table 3.5). The leg kick contributes 78 % of the total net propulsive forces on the 1<sup>st</sup> lap and 79 % on the 4<sup>th</sup> lap (Table 3.5). This demonstrates the heavy reliance on the legs in breaststroke swimming comparable to previous work by Van Tilborgh, et al., (1988) who reported that the legs were responsible for up to 75 % of total impulse during the breaststroke stroke cycle.

Propulsive forces have been shown to be linked to peak velocity (Vilas-Boas, Fernandes and Barbosa, 2010) 2010). It has been previously reported (Sharp, 1986) that CSS and strength measures should be investigated. The results of the current study show that there is no significant relationship ( $p > 0.05$ ) between overall CSS and dry land strength of the legs and arms when strength tests are corrected for body mass. This is what would be expected as previous studies (Vorontsov, 2011) have shown no direct relationship between dry land strength performance and swimming performance in front crawl. The reason for the lack of a relationship between dry land strength and CSS is that a swimmer has to overcome hydrodynamic resistive forces by the means of propulsive forces from the movements of the arms and the legs in the water. The magnitude of these forces depends on the movements' velocities and direction, position of distal arm and leg segments relative to the direction of pushing and pulling actions and the direction of locomotion (Vorontsov, 2011). This indicates that the application of propulsive forces in swimming are specific and complex in nature identifying complex technical skills that are required to achieve maximal propulsive forces. This helps to explain why there is no direct relationship between the dry land strength indices and the participant's performance. This does not mean that strength qualities are not important

characteristics of a breaststroke swimmer, it means that strength testing needs to be more specific to better assess the ability to generate force in the water.

The significant change in CSS with lap coincides with the significant decrease ( $p = 0.035$ ) in the minimal velocity experienced during the arm and leg recovery phases of the stroke cycle. This indicates that participants are experiencing greater hydrodynamic resistive forces during this phase on the 4<sup>th</sup> lap compared to the 1<sup>st</sup> (Table 3.5). The decrease in minimum velocity could have been caused by changes in the body position increasing the frontal surface area of the body therefore increasing the drag forces resulting in a greater loss of linear velocity. However, this idea is not supported by the current results as the participant did not experience an increase in the mean resistive force during the arm and leg recovery phase (phase 4, Figure 3.3) and there was no significant change in arm-leg co-ordination did ( $p > 0.05$ ) or in the time spent in each of the phases of stroke cycle (Table 3.6 and 3.7). The participants were unable to increase their propulsive forces to accommodate for this decrease in linear velocity. The peak velocity achieved during the leg kick did not significantly change ( $p > 0.05$ ) with lap (Table 3.4) indicating that the participants were able to generate similar amounts of force during this phase of the stroke cycle but this may have been at a greater energy cost as there was a significant increase ( $p < 0.05$ ) in the participants heart rate, BLa and following the end of the swim (Table 3.1).

There was no significant change in CSS (Table 3.3) with lap and no significant change in net the propulsive forces (Table 3.6) and peak velocities during phase 1 and 3 (Table 3.4) suggests that the participants were not affected by fatigued, which has been defined as a decrease in the ability to maintain effective mechanical power output (Toussaint et al., 1988) as a result of a decrease in the

effective application of force (Maglischo, Maglischo and Santos, 1987; Changalar and Brown, 1992). However the results of the strength indices and BLa suggest that the participants were fatigued. It would have been expected that fatigue would cause changes in the mechanical efficiency as net propulsive forces would be reduced as the participants progress through the 100 m swim, due to a decrease in ability to generate muscular force (Bigland-Ritchie and Woods, 1984). However this is not supported as there was no significant change in SI in the current study. In contrast previous studies have shown that swimmers try and counteract this loss of mechanical efficiency by increasing the time spent in the propulsive phases of the stroke in front crawl swimming (Schnitzler et al., 2010). The differences in the study could be due to the fundamental differences in the use of the arms and legs in the breaststroke compared to front crawl. Therefore we reject the hypothesis that as the swimmers progressed through the swim there would be a decrease in intra-cyclic velocity fluctuations.

The results of the strength indices show that there is no significant relationship between the dry land strength scores, of the upper body and the legs, and the propulsive forces, of the legs and arms. This is not unexpected as the application of propulsive forces during swimming requires specific and complex technical skills, which may explain why different manifestations of dry land strength are not always directly related to swimming performance (Voronstov, 2011). The assessment of strength in the current study used isometric measurements of the grip and the legs which are being related to dynamic movements that are dynamic and highly technical components of the breaststroke stroke cycle. To better assess strength and skill it may be better to assess the stroke kinematics with simulation force during a tethered assessment.

As the results of the strength test show a significant decrease ( $p < 0.05$ ) from rest to post swim it could be assumed that the participants were less able to produce muscle force and therefore maintain power output, as the swim progressed, and therefore propelling efficiency was reduced as a result of fatigue. Physical fatigue causes a diminishing effect on the execution of motor activities making it harder to maintain stroke length and stroke rate as there is a decrease in stroke efficiency. Although the changes in SL, SR and SI in the current study were not significant (Table 3.2), significant changes in SL and SR have previously been reported in breaststroke swimming (Thompson, Haljand and MacLaren, 2000). The finding of the current study could have been due to the number of participants used in the study which will reduce the power of the statistical analysis. Also in the current study there was a range of abilities in the participants which can be seen in the high inter individual variability in CSS, SR and SL. Physical fatigue has been defined as any reversible decrease in performance (Bigland-Ritchie and Woods, 1984). The reduction in force or power generating capacity is often interpreted as the focal reason for the drop-off in performance. Previous studies (Forestier and Nougier, 1998) have shown that physical fatigue can lead to compensatory mechanisms. Compensatory mechanisms of fatigue are that other muscles take over the function of the muscles that usually perform the repetitive task (Forestier and Nougier, 1998). The current study cannot speculate on likely potential changes in muscle recruitment, but this area warrants further investigation in the future. To better understand the compensatory mechanisms that occur during the stroke cycle we need to understand the neuromuscular changes that are taking place within the muscles involved in the leg kick and the arm stroke. A better understanding could be achieved by looking at surface electromyography (sEMG) and looking at the muscle activation patterns of the key muscles to provide more detailed insight as to why swimmers are not able to

maintain mechanical efficiency and therefore experience a detrimental reduction in CSS over the duration of the swim.

### **3.6 Conclusions**

The findings of the current study demonstrate that as the participants progress through the 100 m maximal breaststroke swim there was a statistically significant change in clean swim speed. Although there was a significant change in clean swim speed there were no significant changes in SR, SL, SI and the co-ordination or timings of the phases of the stroke cycle. Even though there was a significant decrease in the force generating capacity of the arms and legs on the strength indicates these were not related to the peak velocity and/or mean propulsive forces of the arms and legs. These findings suggest that the participants were able to complete the swim using the same timings and spending the same time in each of the stroke cycle phases.

## **CHAPTER 4**

### **CHANGES IN MUSCLE BURST ACTIVITY DURING A 100-M LONG COURSE BREASTSTROKE SWIM**

## CHAPTER 4

### 4.0 Changes in Muscle Activity during a 100-m Long Course Breaststroke Swim

#### 4.1 Abstract

The purpose of the study was to investigate changes in the amplitude and duration of muscle activation of key upper and lower extremity muscles during a 100 m maximum effort swim from a water start in seven specialist breaststroke swimmers; FINA points  $600 \pm 74$ . Surface electromyography (sEMG) was collected from the pectoralis major, latissimus dorsi, biceps femoris, rectus femoris, vastus lateralis and vastus medialis muscles. Kinematic analysis (Stroke rate, stroke length and clean swim speed) and co-ordination analysis was carried out on the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of the swim. Clean swim speed and stroke length significantly decreased from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m ( $p < 0.05$ ). The cycle time (the time between two cycles of muscle activity) did not significantly change ( $p = .123$ ) over the 100 m swim. However, the duration of the active phase expressed as a percentage of cycle time significantly ( $p = .002$ ) decreased over the four 25 m sections of a 100 m swim. The duration of the non-active phase of the muscles significantly increased ( $p = .0001$ ) over the four 25 m sections. The peak sEMG amplitude showed statistically significant effect for muscle ( $p = .002$ ). The only muscle that showed a statistically significantly changed in peak sEMG from the 1<sup>st</sup> to the 4<sup>th</sup> 25 m section was the pectoralis major muscle. It was concluded the most of the muscles studied were working sub maximally throughout the swim apart from a heavy reliance on the pectoralis major muscle which resulted in the accumulation of fatigue in this particular muscle.

## 4.2 Introduction

One of the factors that determine performance in swimming is the ability of the central nervous system (CNS) to co-ordinate the activation and deactivation dynamics of muscle force development and relaxation (Billaut, Basset and Falgairette, 2005) and therefore to optimise muscle work and co-ordination. The understanding of how motor patterns (muscle activation, timing and magnitude) are performed is one of the key areas of research in biomechanics. Little is known about the neural basis of movement control strategies and whether strategies are the same across movements, or are unique and task dependent. In front crawl swimming stroke rate (SR) and swimming velocity are the two main control parameters that determine co-ordination (Seifert, Chollet and Chatard, 2007). However, it has been reported (Seifert et al., 2009) that some swimmers don't change their co-ordination through speed but only change SR. In breaststroke swimming for a given speed individuals will adopt a particular combination of stroke rate (SR) and stroke length (SL). This combination will at least partly be determined with the individual co-ordination pattern that is utilised by that swimmer. In competitive breaststroke swimming three distinct styles of co-ordination have been observed, *Glide*, *Continuous*, and *Overlap* (Maglischo, 2003; Oxford et al., 2010) (Chapter 1). The combination of SR, SL (Thompson, Haljand and MacLaren, 2000) and the timing between the propulsive phases of the arms and legs (Oxford et al., 2010) have been shown to change as the race progresses.

Muscle fatigue can be defined as the failure to maintain a required or expected power output (Edwards, 1983). However, the maximal force generating capacity of the muscles starts to decline progressively during exercise indicating that the onset of fatigue begins before the



muscles fail to perform the required task (Gandevia, 2001). In swimming, efficiency is discussed as gross or mechanical efficiency ( $\eta_o$ ) (Toussaint et al., 1988; Toussaint, Janssen and Kluft, 1991; Zamparo, 2006; Zamparo et al., 2008) which is the efficiency with which the metabolic power input ( $\dot{E}$ ) is transformed into useful mechanical power output (See section 1.5.1 of the literature review for more detailed discussion). Movement in swimming is an energetically costly activity with values of drag efficiency ranging from 0.03 - 0.09 (di Prampero et al., 1974; Holmer, 1972; Pendergast et al., 1977; Toussaint et al., 1988). Thus, indicating that less than 10% of the mechanical power output can be transformed into useful mechanical power to overcome drag forces. Mechanical power production and muscle efficiency is dependent on the rate at which muscle shortens (Wakeling, Blake and Chan, 2010). Maximal power output of a muscle occurs while they are maximally active and shorten at their 'optimal speed' for power production. Optimal speed for a contracting muscle is about 25-36 % of their maximal intrinsic speed (Josephson, 1999; He et al., 2000) which overlaps with the range of speeds at which maximal muscle efficiency has been shown to occur, 15 - 29 % of maximal intrinsic speed (He et al., 2000).

During dynamic movements it is not physiologically possible or efficient for each muscle to work at maximal power output (Wakeling, Blake and Chan, 2010). Maximal muscle power outputs occur when the muscle is both fully activated and shortening at optimal speed for power generation; however muscles are seldom fully activated and anatomical constraints may prevent the muscle from shortening at this optimal speed due to fibre pennation angles and tendon compliances (Lieber and Friden, 2000). During dynamic joint movements the moment arms are continuously changing with multiple muscles around the joint involved, meaning that muscles

may not shorten at their optimal speeds (Wakeling, Blake and Chan, 2010). However optimal angular velocities have been shown to occur with maximal joint power in cyclic contractions (Sargeant, Hoinville and Young, 1981; Zoladz, Rademaker and Sargeant, 2000). Muscles may contribute to a range of functions during a movement, such that muscles that span a single joint (mono-articular) may be important for producing power, (Janshen, Mattes and Tidow, 2009) whereas a muscle that spans multiple joints (bi-articular) could be more important for controlling the external force (Van Ingen Schenau et al., 1992). Activity of a biarticular muscle may produce joint movements in a useful direction at one joint but in paradoxically inappropriate direction in the other (Lombard, 1903). This is known as Lombard's paradox and has the potential to decrease efficiency in a biarticular muscle compared to a mono-articular muscle (Gregor, Cavanagh and LaFortune, 1985). Biarticular muscles are reported to play a role in the transfer of energy between joints and also in the control of the direction of force (Van Ingen Schenau et al., 1992).

In breaststroke, the quadriceps muscles and the upper body muscles pectoralis major, latissimus dorsi and deltoids (see Table 1.2 in Chapter One for kinesiological analysis of the breaststroke) have been shown to play a major part during the breaststroke cycle. These muscles are either involved in the movement of the limbs into a position to be able to apply force or during the application of force to the water (Rodeo, 1984). In breaststroke swimming there are two periods where forward velocity increases, with one increase occurring during the leg kick and the second during the arm pull phases of the stroke (Leblanc et al., 2007). Breaststroke swimmers have been categorised as either arm dominant or leg dominant depending on the phase in which peak velocity occurs (Leblanc et al., 2007).

The vastus lateralis and vastus medialis are both mono-articular muscles that play an important role in power production during extension of the knee joint (Janshen, Mattes and Tidow, 2009; Ryan and Gregor, 1992; Wilson, Robertson and Stothard, 1988; Yoshizawa et al., 1976). The rectus femoris is a biarticular muscle which is also used for extension of the knee joint. The knee flexor and hip extensor muscle biceps femoris is a bi-articular muscle that is active during the end of knee extension and also plays a role in hip extension (Hug and Dorel, 2009). In the early part of the leg kick the muscles vastus medialis and rectus femoris have been shown (Tokuyama, Okamoto and Kumamoto, 1976; Yoshizawa et al., 1976; Yoshizawa et al., 1978) to exhibit high levels of activity while extending the knee to produce propulsion in skilled adult swimmers compared to unskilled children who showed limited activity of these muscles (Tokuyama, Okamoto and Kumamoto, 1976). The biceps femoris has been shown to be active during the hyperextension of the hip joint which corresponded to the arm pull phase (Tokuyama, Okamoto and Kumamoto, 1976) in skilled swimmers. However in unskilled swimmers the rectus femoris showed noticeable activity during this phase as the thighs were shown to be in a flexed position (Tokuyama, Okamoto and Kumamoto, 1976). This flexing of the thighs places the legs in a position that increases the drag on the swimmer thus decreasing linear velocity. In the first part of the glide phase, high levels of activity of the vastus medialis and the rectus femoris have been reported (Yoshizawa et al., 1976; Yoshizawa et al., 1978) with reduced levels of activity in the latter part of the glide phase in skilled swimmers. Less skilled swimmers have reported lower levels of activity in these muscles during this phase (Yoshizawa et al., 1976). The lower levels of activity in the lower skilled swimmers were attributed to the lack of full extension of the knee joint (Yoshizawa et al., 1976). The posterior deltoid muscle was shown to have high levels of activity throughout the glide phase of the stroke (Yoshizawa et al., 1976). Yoshizawa,

et al., (1979) reported that there were higher levels of muscle activity in the latter phases of the glide for the posterior deltoids as they are active in the extension and outward rotation of the glenohumeral joint.

The posterior deltoid has been shown to be the most active of the three heads of the deltoid muscle during the whole stroke with peak activity occurring during the early pull through phase (Ruwe et al., 1994; Tokuyama, Okamoto and Kumamoto, 1976). . During the arm pull phase of the stroke cycle the pectoralis major and latissimus dorsi muscles work together in the extension, adduction and internal rotation of the glenohumeral joint. The pectoralis major muscle has been reported to produce high levels of activity throughout the arm recovery phase and at the beginning and middle of the arm pull phase (Nuber et al., 1986). The latissimus dorsi muscle has been shown to demonstrate activity during the pull phase of the arms with peak activity at the midpoint of the arm pull phase (Nuber et al., 1986; Tokuyama, Okamoto and Kumamoto, 1976). The biceps brachii muscle has been reported to show no distinct pattern of activation throughout the whole stroke with low activity throughout the stroke (Nuber et al., 1986). The triceps brachii muscle is used to fully extend the elbow during the extension of the arms during the arm recovery phase (Nuber et al., 1986). Co-contraction of the biceps brachii and the triceps brachii has been reported (Yoshizawa et al., 1976) during the pull phase due to active flexion of the forearm whilst the elbow joint is stabilised.

Yoshizawa et al., (1976) investigated activity during breaststroke and found there is continuous activity of the biceps femoris through out the recovery phase of the leg kick. Previous studies (Yoshizawa et al., 1976; Yoshizawa et al., 1978) have only looked at activity in the glide

and the continuous co-ordination patterns, but have not addressed the activity during the overlap co-ordination pattern of the breaststroke. Further analysis to understand the difference between the overlap technique and the glide technique would increase the level of knowledge of the co-ordination pattern of the overlap swimmers. The other limitation of the previous studies is that they have not investigated how muscle activity changes during a swim, as swimmers have been shown to change the timing of the arms and legs (Oxford et al., 2010) as they progress through the swim. Previous studies have shown a decrease in clean swim speed with subsequent changes in stroke rate (SR) and stroke length (SL) (Oxford et al., 2010; Takagi et al., 2004; Thompson, Haljand and MacLaren, 2000) as swimmers progress through a swim and changes in the swimmers ability to maintain force production in the arms and the legs following a 100 m maximal effort swim (Chapter 3). The decrease in clean swim speed observed in breaststroke competition (Thompson, Haljand and MacLaren, 2000) and in testing (Oxford et al., 2010) has been associated with changes in the stroke parameters SR and SL (Thompson, Haljand and MacLaren, 2000). Thompson, et al., (2000) showed a decrease in clean swim speed was associated with a decrease in SL and SR. It has been reported (Pai, Hay and Wilson, 1984) that a decrease in SR could be associated with a compensatory mechanism. The decrease in SR could also reflect a diminution of neural activation (Keskinen, 1994), this has been partially validated Caty, et al., (2007) who reported a decrease in the EMG frequency of the forearm muscles during a 4\*50 m maximal intensity front crawl swims. Further quantification of the progression of muscle fatigue could be a valuable tool in the development of specific training protocols (Rouard, 2011). Even though there are a number of studies that have looked at co-ordination in breaststroke swimming (Chollet et al., 2004; Leblanc et al., 2007; Seifert and Chollet, 2005), none have investigated how it relates to changes in muscle activity.

In the evaluation of co-ordination and muscle fatigue, the use of surface electromyography (sEMG) is commonly used (Bobbert and van Ingen Schenau, 1988; Hautier et al., 2000; Van Ingen Schenau et al., 1992; Strin et al., 2011). It is well known that inter-muscular co-ordination is modified during dynamic movements due to fatigue (Prilutsky, 2000; Van Ingen Schenau et al., 1992). Muscular co-ordination is defined as “a distribution of muscle activation or force among individual muscles to produce a given combination of joint moments” (Prilutsky, 2000). With fatigue occurrence, the time delay between the surface sEMG onset of the agonist and antagonist muscles changes, affecting activation and deactivation dynamics, reflecting changes in the motor pattern. Psek & Cafarelli, (1993) examined the activation of antagonistic muscles under fatigue conditions and reported that the fatigue of the vastus lateralis increased the amplitude of activation of the biceps femoris which acts as an antagonist in knee extension movements. The proposed mechanism for this increase was the common drive hypothesis. This hypothesis suggests that the central nervous system may control each muscle’s motor neuron pool by a single input when both muscles are participating in a task (DeLuca and Mambrito, 1987). This suggests that fatigue of a muscle group decreases the overall efficiency of a movement by de-organising muscular co-ordination (Hautier et al., 2000). The effects of fatigue in swimming have been reported for the rotator muscles of the shoulder (Monteil et al., 1996). Fine wire EMG electrodes were used at the beginning and end of a 400 m front crawl test in a flume. In the fresh condition high specificity of the internal and external muscles, infraspinatus, teres minor and supraspinatus respectively, to the stroke cycle was reported. In the fatigued state a loss of specificity was reported for the internal and external rotator muscles. From the fresh to the fatigued condition there was an increase in the EMG’s (IEMG) amplitudes (Monteil et al., 1996). Similar increases in amplitude were reported by (Wakayoshi et al., 1994) for the

deltoideus and the flexor carpi radialis when participants swam to exhaustion in a flume. The greater EMG's amplitudes indicate that the participants were performing at sub maximal levels when fresh as there was an increase in the motor unit recruitment (Rouard, 2011). However a study by Rouard and co-workers (Rouard, Quezel and Billat, 1992) analysed six upper body muscles during a 4\*100 m with 45 s rest intervals in front crawl and observed no change in iEMG for any of the muscles apart from the flexor carpi ulnaris muscle which showed a significant increase in amplitude. It was concluded that no change indicated a sub-maximal effort when fresh that allowed the muscle to maintain performance throughout the swim. A study by Caty et al., (2007) reported a significant percentage decrease in the mean frequency (MNF) of the power spectral density of the flexor and extensor carpi ulnaris during 4\*50 m front crawl swims with 10 s rest. The fatigue of these muscles decreased the ability to fix the wrist thus reducing forward propulsion. In all these studies inter-individual differences were reported which, it is suggested, reflect individual variability in muscle fibre and/or motor unit recruitment (Caty et al., 2007).

To better understand the changes in co-ordination as a result of the fatigue process during the breaststroke, the role of individual muscles should be considered. The use of sEMG enables the simultaneous monitoring of the muscle activity that potentially can provide greater understanding of the fatigue process (De Luca, 1984; Masuda et al., 1999). The amplitude of the sEMG during sustained muscle contractions has been shown to increase (Masuda et al., 1999; Merletti, Lo Conte and Orizio, 1991). One reason for the increase in muscle amplitude is due to an increased synchronisation of the recruited motor units (MU) and recruitment of new MU's (Masuda et al., 1999; Merletti, Lo Conte and Orizio, 1991). For instance a study by Wakayoshi,

et al., (1994) reported no change in the degree of muscle fatigue in the flexor carpi radialis, biceps brachii, triceps brachii and deltoid muscles at various swimming velocities in front crawl swimming. However, only the deltoid muscle showed an increase in integrated EMG at swim velocities between 1.3 and 1.4 m·s<sup>-1</sup> whilst the other muscles showed no significant change.

Inter-muscular co-ordination patterns can be described by the activation and deactivation dynamics that represent the delay between muscle force development and relaxation (Billaut, Basset and Falgairette, 2005). Little is known about the changes of the active and non-active phase timing of muscle activity during a swim. The active phase of the stroke has been defined (Baum and Li, 2003; Strin et al., 2011) as the part of the sEMG signal that is above 10% of the local maximum energy value for a particular burst of muscle activity. The non-active phase has been defined as the part of the sEMG signal that is below the 10 % local maximum energy value. A study by Strin, et al., (2011) investigated the changes in the pectoralis major (upper and lower), latissimus dorsi (upper and lower), biceps brachii and triceps brachii local maximum energy values during a 100 m maximal effort front crawl swim. The authors reported a significantly shorter duration of the active phase in the pectoralis major muscle ( $p < 0.05$ ) compared to the other muscles. The non-active phase of the pectoralis major was significantly longer ( $p < 0.05$ ) than the non-active phase of the muscle triceps brachii. Strin, et al., (2011) also reported a significant increase ( $p < 0.05$ ) in the AF of the muscle latissimus dorsi (lower part) from the beginning of the swim to the end of the swim from  $387 \pm 49$  to  $425 \pm 72$  ms. The results also showed that from the beginning to the end of the swim there was a significant increase ( $p < 0.05$ ) in the non-active phase in all the muscles studied. These findings suggest that compensatory mechanisms with respect to a shift in the level of activity between muscles are



being used to try and maintain swim speed. Ikuta, et al., (2012) reported compensatory mechanisms in 200 m front crawl swimmers between the pectoralis major and other muscles. Previous research in cycling (Wakeling, Blake and Chan, 2010) has shown compensatory mechanisms in the lower-limb muscles with Szucs, et al., (2009) reporting compensatory mechanisms in the upper body muscles during a push-up task.

There are no studies that have investigated the changes in inter-muscular co-ordination during a breaststroke swim. The current investigation of muscle activity and inter-muscular co-ordination will help clarify the findings of previous research (Oxford, et al., 2010; Takagi, et al., 2004; Chapter 2) which have reported changes in co-ordination of the arms and legs as a result of fatigue. It is important to know whether the decreases in force (Aujouannet et al., 2006) and power production (Toussaint et al., 2006) that accompanies fatigue is completely attributable to contractile performance loss or whether changes in muscle activation accounts for a part of this loss (Hautier et al., 2000). This new research will provide further insight into how the body co-ordinates movements and adapts to the demands of the 100 m swim. This information is useful in many fields such as analysis of performance requirements, injury prevention and rehabilitation strategies. EMG analysis is a useful tool to individualise strengthening and endurance muscular training processes and to prevent muscle imbalances between muscle groups (Rouard, 2011).

#### 4.2.1 *Aims*

To investigate the changes in amplitude of muscle activity between the 1<sup>st</sup>, 2<sup>nd</sup>, 3<sup>rd</sup> and 4<sup>th</sup> 25 m sections of a 100 m breaststroke swim.

To determine whether there are changes in the durations of the active and non-active phases of the muscle during the 1<sup>st</sup>, 2<sup>nd</sup>, 3<sup>rd</sup>, and 4<sup>th</sup> sections of a 100 m breaststroke swim.

To investigate changes in kinematic parameters swim speed, stroke rate and stroke length between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m breaststroke swim.

To investigate the changes in co-ordination of the arms and the legs in the between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m breaststroke swim

#### 4.2.2 *Hypothesis*

- 1) It was hypothesised that there will be a decrease in the peak amplitude of the muscles from the 1<sup>st</sup> to the 4<sup>th</sup> 25 m section of the 100 m breaststroke swim.
- 2) It was hypothesised that there will be a decrease in the active phase and a subsequent increase in the non-active phase of the muscle from the 1<sup>st</sup> to the 4<sup>th</sup> 25 m section of the 100 m breaststroke swim.

- 3) It was hypothesised that there will be a decrease in clean swim speed with subsequent changes in stroke length and stroke rate from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m of the 100 m breaststroke swim.
- 4) It was hypothesised that there will be a change in the timing of the co-ordination phase from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m of the 100 m breaststroke swim.

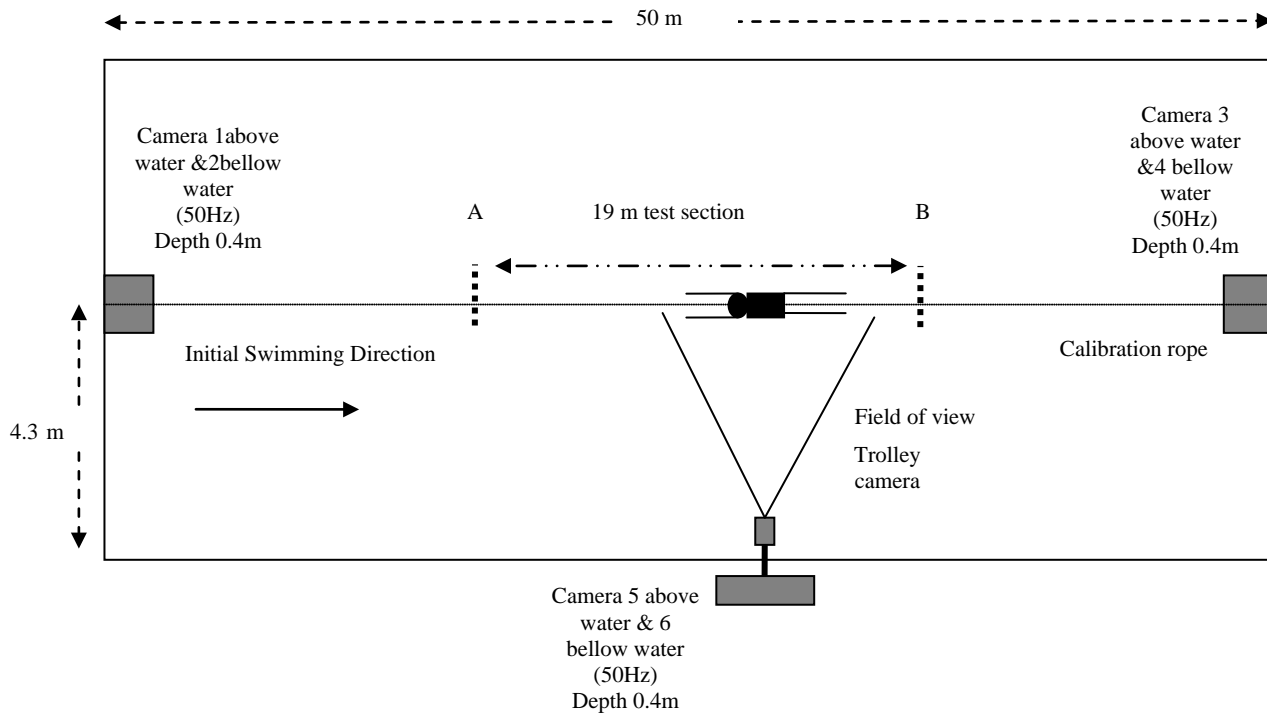
## **4.3 Methods**

### *4.3.1 Participants*

Participants were seven male competitive breaststroke swimmers, aged  $19.8 \pm 2.1$  years, body mass  $75.6 \pm 3.6$  kg, height  $1.81 \pm 0.60$  m. All participants were members of the ASA National Beacon Squad, FINA points  $600 \pm 74$  based on FINA points scoring 2012 for 100 m long course for breaststroke (Splash; FINA point calculator; GeoLogix, Bern, Switzerland). The swimmers' regular weekly training schedule consisted of approximately 12-20 h of swimming along with 2-4 h of dry land training. The participants were asked to refrain from heavy strength training 48 h prior to the study. The procedures and the purpose of the study were fully explained to participants and they all signed the informed consent forms. The study was approved by the Coventry University Ethics Committee.

#### 4.3.2 Testing Procedure

All testing took place in the same 50 m pool at the same time of day during morning swimming between the hours of 6 and 8am. For sEMG analysis the pool was split up into 25 m sections (Figure 4.2). Once the participants had prepared for EMG data collection they completed a 500 meter warm up. The warm up consisted of a series of ten 50 m swims at medium level effort (Strin et al., 2011). After the warm up the participants were instructed to complete a 100 m breaststroke maximal effort swim, with no race strategies, from a water start.



**Figure 4.1** A plan view of the filming set-up used for qualitative analysis.

### 4.3.3 *Swim Trials*

Time to complete 100 m was recorded using a hand held stop watch (Fastime 7, AST, Leicestershire, UK) as the time from when the feet left the wall at the start until the double hand touch on the wall at the end of the swim.

Five digital video cameras (Sony video HDR-HC9, Sony, Tokyo, Japan), sampling at 50Hz with a shutter speed of 1/300 s were used to film the participants above and below the water. One camera was placed above the water at a height of 0.6 m and one camera, enclosed in a custom made waterproof housing (Plate 1.0; Chapter 2), was placed below the water at a depth of 0.4 m, at each end of the lane to record frontal and rear views (Figure 4.1). The fifth camera (Sony video HDR-HC9, Sony, Tokyo, Japan) was attached above water on the trolley. The field of view was adjusted on all cameras so that the whole body of the participant was visible (Figure 4.1). A sixth underwater camera, a waterproof bullet camera, connected to a visual display unit (VDU; Sony digital video cassette recorder GV-D800E, Sony, Tokyo, Japan) was attached to a trolley (Figure 2.0, Chapter 2, Section 2.3.2). The frontal and rear views of the participant were synchronised to the sagittal view (Qunitic, V17, Warwick, UK) using a custom made LED light trigger system that could be seen in all six camera views. The trolley was manually moved at the same speed as the participants, parallel to the greater femoral trochanter to maintain the joint marker in the approximate centre of the field of view (Figure 4.1), in the sagittal plane, throughout the entire 100 m swim. Refraction due to the water was not accounted for in this study as refraction in water due to light rays is relatively low, with previously reported values that vary between 0.39 and 1.28 % (Gourgoulis et al., 2008).

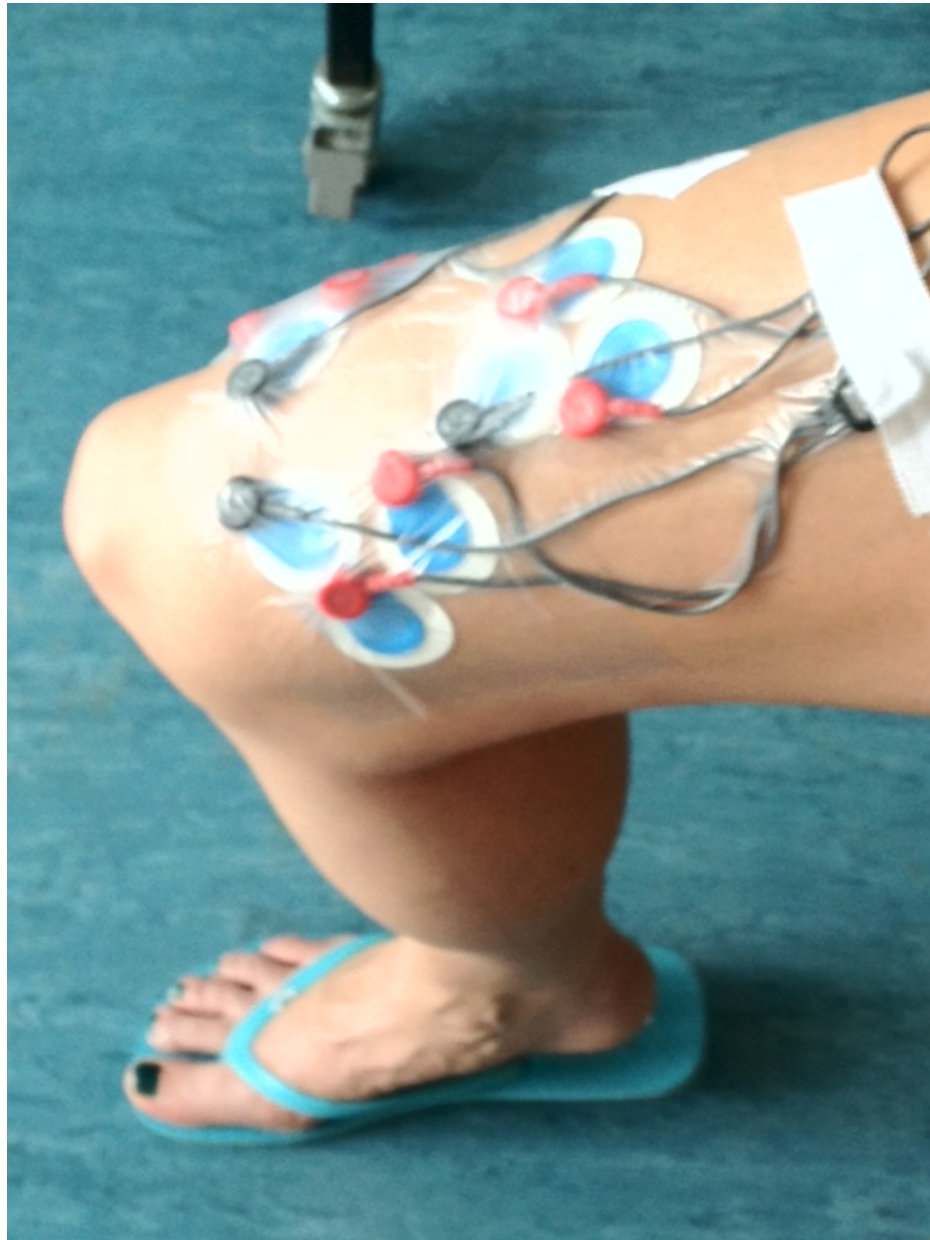
#### 4.3.4 Stroke Parameters

The following stroke parameters were calculated from the 19 m testing sections of the pool on each of the two laps of a 100 m swim from the sagittal view video recordings (trolley camera, Figure 4.1). *Clean Swim speed* ( $\text{m}\cdot\text{s}^{-1}$ ) was defined as the mean forward speed of the greater trochanter over the 19 m testing section on each lap (Figure 4.1) monitored from the time the greater trochanter entered the 19 m testing section until it left the 19 m testing section (Figure 4.1) via the stop clock located on the video analysis software (Qunitic, V17, Warwick, UK) (to the nearest  $0.01 \text{ m}\cdot\text{s}^{-1}$ ); *Stroke frequency* ( $\text{stroke}\cdot\text{min}^{-1}$ ) was defined as the number of stroke cycles performed in one minute, calculated for each 19 m testing section (Figure 2.1) (to the nearest  $0.01 \text{ strokes}\cdot\text{min}^{-1}$ ); *Stroke length* ( $\text{m}\cdot\text{cycle}^{-1}$ ) was defined as the distance that the participants greater trochanter travelled in one stroke cycle, computed from the clean swim speed and the stroke frequency values (to the nearest  $0.01\text{m}$ ) (Equation 2.1, Section 2.3.5); *Stroke Cycle Time* (s) was defined as the time taken to complete one complete stroke cycle, calculated as the mean stroke cycle time over each 19 m testing section (to the nearest  $0.02 \text{ s}$ ); Co-ordination phase 1 (CPhase1) (Chapter 2) was calculated as the time between the end of the leg kick phase and the start of the arm recovery phase and was used to classify each participant's co-ordination as overlap (represented by a negative value to the nearest  $0.02 \text{ s}$  indicating simultaneous propulsion of the arms and legs), glide (represented by a positive value to the nearest  $0.02 \text{ s}$  indicating a delay (glide) in the initiation of the arm pull phase) or continuous.

#### 4.3.5 *Collection of the EMG Data*

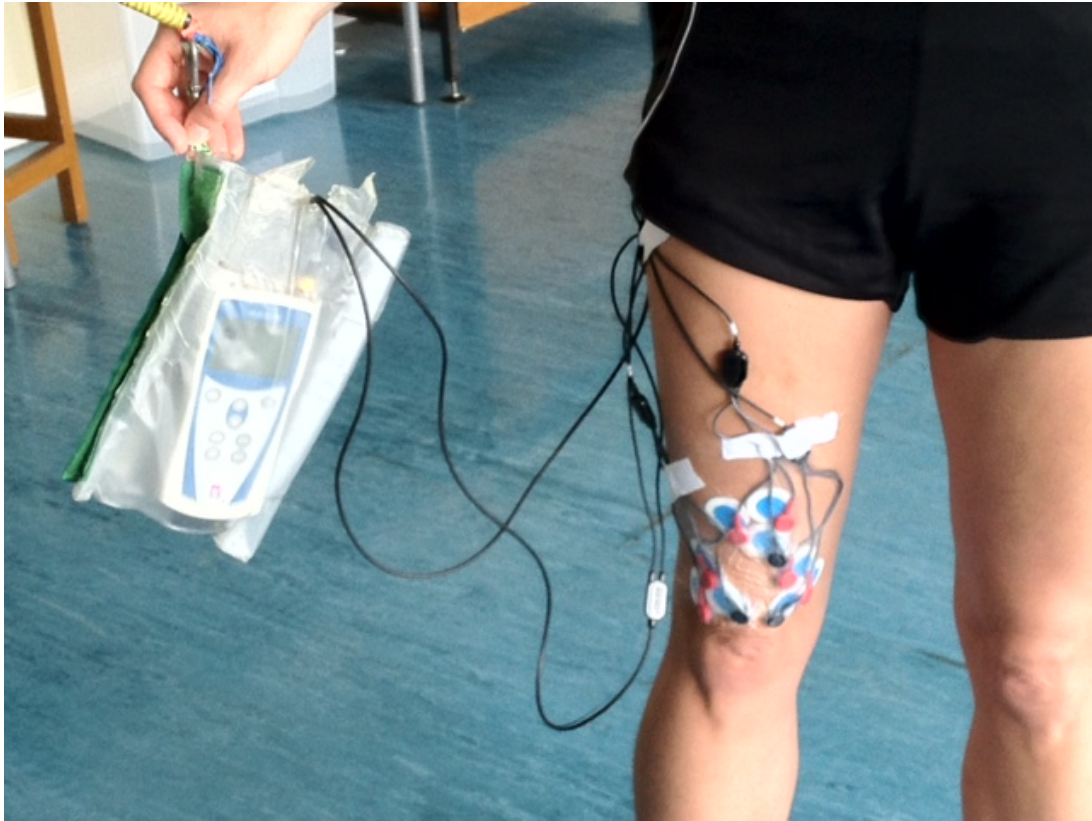
Passive bipolar surface electrodes of 30mm diameter (Blue Sensor, Ltd, Denmark, Ag/AgCl) were used to measure muscle activation of the pectoralis major, latissimus dorsi, anterior deltoid, bicep brachii, tricep brachii, rectus femoris, vastus lateralis, vastus medialis and bicep femoris. The Surface electrodes were placed on the contracted belly of each of the muscles, in line with the muscle fibre direction in accordance with recommendations of SENIAM (Surface EMG for Non-Invasive Assessment of Muscles) (Hermens and Freriks, 1997) with an inter electrode distance of 1.5cm (Basmajian, 1973; Caty et al., 2007). Before electrode placement, the participant's skin was prepared in accordance with International Society of Electromyography and Kinesiology (ISEK); the area was shaved, in order to reduce skin impedance and cleaned with alcohol wipes. The electrodes were covered with a waterproof transparent dressing to prevent water damage to the electrodes (Rouard & Clarys, 1995; Strin, et al., 2011; Plate 4.1). Vaseline was applied to the edges of the dressing to ensure waterproofing. Adhesive elastoplast tape was used to fix the cables to the skin, to avoid interference with the signal and the participant whilst swimming (Strin et al., 2011). Electromyography activity was recorded via an ME6000 system (Mega Electronics Ltd., Finland), with an input impedance of less than  $10^{15}/0.2$  ohm/pF, a common mode rejection ratio at 60 Hz of greater than 110dB, a noise level of 1.2 mV, a gain of  $10 \pm 2\%$  and a bandwidth range from 0 Hz – 500 Hz. Muscle activity was sampled at 1000 Hz via a 16bit DAQ-516 A/D card and stored on a laptop computer using MegaWin software (Mega Electronics Ltd., Finland). The telemetric EMG device (MEGA ME6000) was encased in a custom made water proof housing (Plate 4.2) and fixed to a rod. A

research assistant walked along the poolside, next to the participant to keep the EMG device (MEGA ME6000) and cables free of the participants (Plate 4.3).

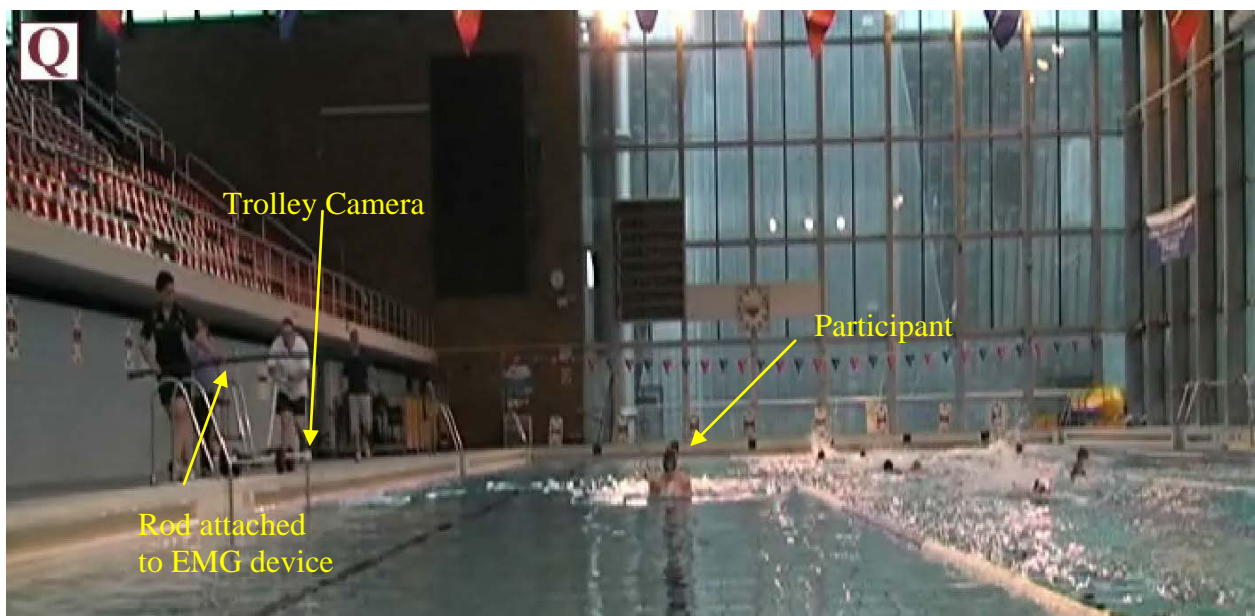


**Plate 4.1** The waterproof dressing used to protect the sEMG electrodes.





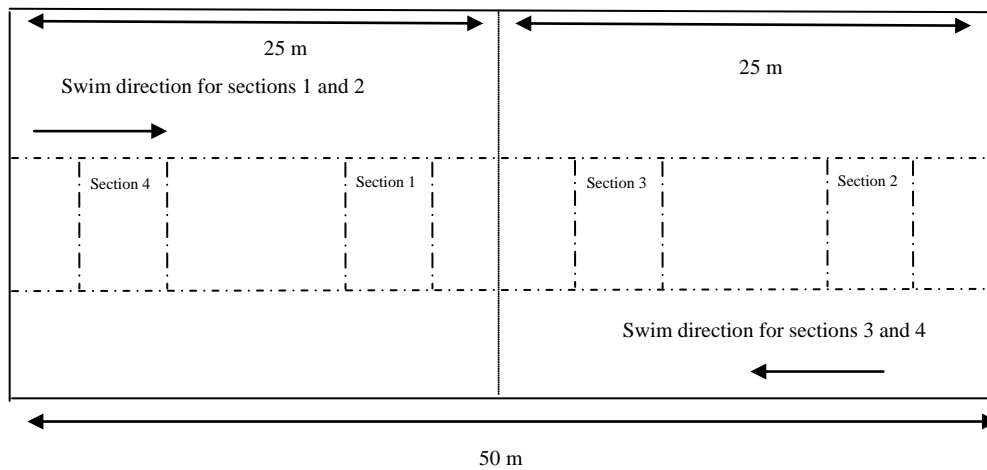
**Plate 4.2** The custom made waterproof housing encasing the MEGA ME6000



**Plate 4.3** The position of the EMG in relation to the trolley camera and the participants

#### 4.3.6 EMG Signal Processing

The raw EMG data were filtered using a high and low pass Butterworth filter (5-500 Hz) and then full-waved rectified. Some EMG electrodes were contaminated with noise due to exposure to water. Any data that were influenced by apparent noise (about  $\geq 3$  SD) was discarded (Ikuta et al., 2012).

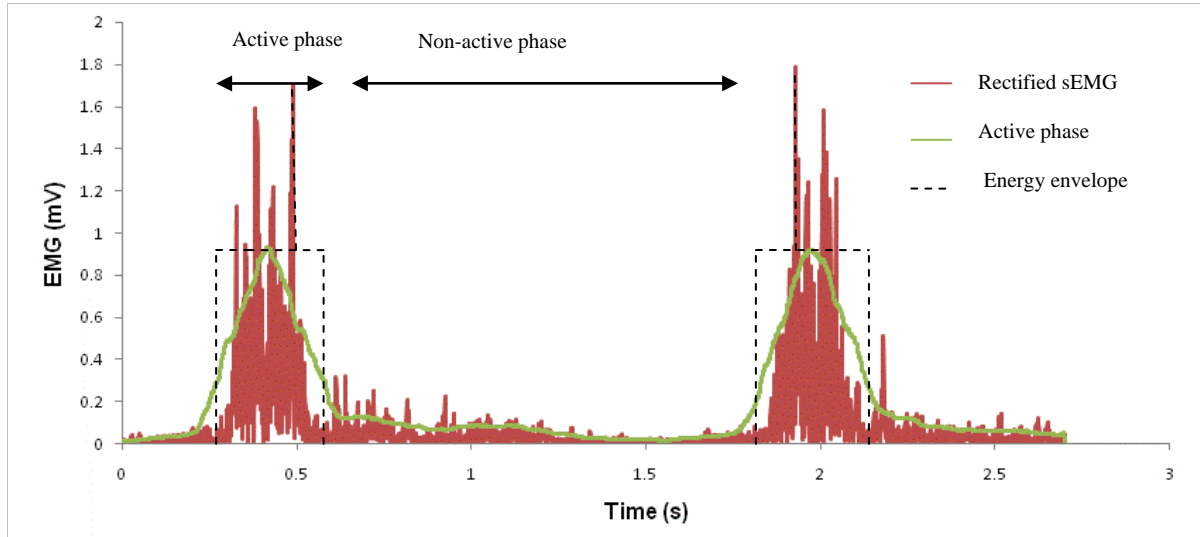


**Figure 4.2** The four sections of the pool where sEMG signals were collected for three complete strokes per section.

To obtain the amplitude and frequency description of the sEMG signal for each swimming stroke, the active phase of the muscle was determined individually for every stroke and every muscle. Three strokes were analysed for EMG during each of the four sections of the swim 1<sup>st</sup>, 2<sup>nd</sup>, 3<sup>rd</sup> and 4<sup>th</sup> section of the 100 m swim (Figure 4.2). Each section was 5 meters from

the end of the subsequent 25 m (Figure 4.2) to avoid the effects of turning and finishing on the sEMG results. First the energy envelope  $E(t)$  of the rectified EMG signal  $x(t)$  was calculated using a sliding data window of 200 ms length in accordance with Strin, et al., (2011).

Muscle activity within each stroke resulted in a local maximum in the energy envelope. For each muscle activation within the stroke cycle the active phase was defined in accordance to Strin, et al., (2011). Initially a threshold value of 10% of the maximum amplitude values across each muscle contraction was used to determine the active phase. The non-active phase was defined as the time interval between two successive active phases (Figure 4.3). Visual inspection was used to determine if 10 % was the appropriate threshold. Appropriate thresholds reflect easily identifiable active phase and non-active phase. If 10 % was not appropriate then the threshold was increased by 5% until an appropriate threshold was determined in accordance with Baum & Li, (2003). Upon reaching the determined threshold the muscle was considered active and the muscle activation duration was defined in units of time. This was done on an individual basis for each phase of muscle activity for each of the contractions to determine the mean muscle activity for each of the four sections of the 100 m swim (Figure 4.3). The average duration of three active phases and the average duration of three non-active phases were calculated for each individual, for each muscle, over each of the four 25 m sections of the swim. The relative duration of the active phase and non-active phase were expressed as a percentage of cycle time (CT). Cycle time was calculated as the sum of the mean active phase and the mean non-active phase over three cycles of muscle activity.



**Figure 4.3** sEMG muscle burst activity as a function of time. The dashed lines indicate the durations of the active phases. A threshold of 20 % of the maximum sEMG has been used in this example.

The peak sEMG amplitude of the envelope was calculated as the peak EMG that occurred during the active phase found within each muscle contraction and was averaged over the three active phases for each muscle, to produce separate values for each of the four 25 m sections for each individual. Peak sEMG amplitude of the envelope were normalised to the highest peak ( $\text{Peak}_{Task}$ ) of the dynamic activity (Ball and Scurr, 2010; Burden, 2010; Clarys et al., 2010) of the twelve muscle contractions analysed over the four sections of the swim Figure 4.2). The mean active sEMG amplitude was calculated as the mean of all the sEMG during each active phase for each muscle over each of the four 25 m sections of the swim as the mean amplitude between the onset and offset time of the active phase, to produce separate values for each of the four 25 m sections for each individual (Figure 4.3).

Muscle activity for a complete stroke cycle (cycle time) was defined as the time from the initial onset time of the rectus femoris muscle to the subsequent onset time of the rectus femoris muscle. The onset time of the rectus femoris was taken as the start of a stroke cycle. The time between the end of the active phase of the rectus femoris to the onset time of the active phase of the upper body muscles pectoralis major and latissimus dorsi was calculated to allow a comparison between of the glide and overlap patterns. The synchronisation of active phase of the lower and upper body muscles for the glide and overlap co-ordination patterns were calculated as an average of three complete muscle cycles during the first 25 m section (Figure 4.2).

#### *4.3.7 Statistical Procedures*

Statistical analyses were conducted using SPSS version 16.0 (SPSS, Inc., Chicago, IL, USA). Standard statistical methods were used to calculate mean and standard deviation for all measured variables. Normal distribution of the data was verified using Levene's test. The percentage data was tested for normal distribution using the  $z$ -score of skewness and kurtosis in accordance with Vincent, (2005). If normal distribution was not reported then data was corrected for heterogeneity of variance using the Arcsine transformation (Black, 1999). Paired sample T tests were used to determine differences within stroke length (SL), stroke rate (SR), clean swim speed (CSS) and co-ordination phase 1 (CPhase 1) from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m of the 100 m swim. The effect size of the independent T test was estimated using Pearson's correlation coefficient in accordance with Rosnow & Rosenthal, (2005), with the resultant values interpreted according to Cohen, (1988) as  $r = 0.10$  (small effect),  $r$

=0.30 (medium effect) and  $r = 0.50$  (large effect) results and reported in accordance with Field (2009). Two-way analysis of variance was used to compare selected kinematic variables at the same point of each of the four sections, with section (1, 2, 3 and 4) and muscles (rectus femoris, biceps femoris, vastus lateralis, vastus medialis, pectoralis major and latissimus dorsi) as the fixed factors. When Mauchley's test of sphericity indicated a minimal level of violation ( $> 0.75$ ) the degrees of freedom was corrected using the Huynh-Feldt adjustment and when the sphericity was  $< 0.75$ , the Greenhouse-Geiser correction was used (Field, 2009). Where differences were noted in ANOVA, pairwise comparisons (Bonferroni adjusted) were employed to identify where the significant differences occurred. One-way analysis of variance with Bonferroni post-hoc tests were used with muscle and 25 m section (1, 2, 3 and 4) as the factors to compare selected variables at the same point of each of the four sections. A level of  $p < 0.05$  was considered statistically significant. The effect size for the ANOVA statistics was estimated using partial Eta squared ( $\eta^2$ ) for analysis of variance. Since this method is likely to overestimate effect sizes, values were interpreted according to Ferguson (2009) as no effect if  $0 \leq \eta^2 < 0.05$ ; a minimum effect if  $0.05 \leq \eta^2 < 0.26$ ; a moderate effect if  $0.26 \leq \eta^2 < 0.64$ ; and a strong effect if  $\eta^2 \geq 0.64$ . All ANOVA results were reported in accordance with Field (2009).

## 4.4 Results

### 4.4.1 Performance Data

**Table 4.1** Mean ( $\pm$  SD) and the coefficient of variation as a percentage (cv %) for the values of clean swim speed, stroke length, and stroke rate, for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m swim.

	1 <sup>st</sup> 50 m	2 <sup>nd</sup> 50 m
Clean Swim Speed ( $\text{m}\cdot\text{s}^{-1}$ )	1.20 $\pm$ 0.03 (cv=3)	1.12 $\pm$ 0.04 (cv=4)*
Stroke Length ( $\text{m}\cdot\text{cycle}^{-1}$ )	1.64 $\pm$ 0.09 (cv=6)	1.51 $\pm$ 0.17 (cv=12)*
Stroke Rate(stroke $\cdot\text{min}^{-1}$ )	44.3 $\pm$ 2.2 (cv=5)	45.3 $\pm$ 4.4 (cv=10)

\*Denotes a statistically significant difference  $p < 0.05$  between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m

Overall there was a statistically significant  $t(6) = 4.74$ ,  $p = 0.003$ ,  $r = 0.89$ , decrease of 6.4 % in CSS from the 1<sup>st</sup> 50 m to the 2<sup>nd</sup> 50 m (Table 4.1). There was a statistically significant  $t(6) = 2.76$ ,  $p = 0.032$ ,  $r = 0.75$ , decrease of 7.9 % in SL from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. There was no change in SR,  $t(6) = -0.91$ ,  $p = 0.28$ ,  $r = 0.35$ , from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m of the swim.

#### 4.4.2 Co-ordination Changes

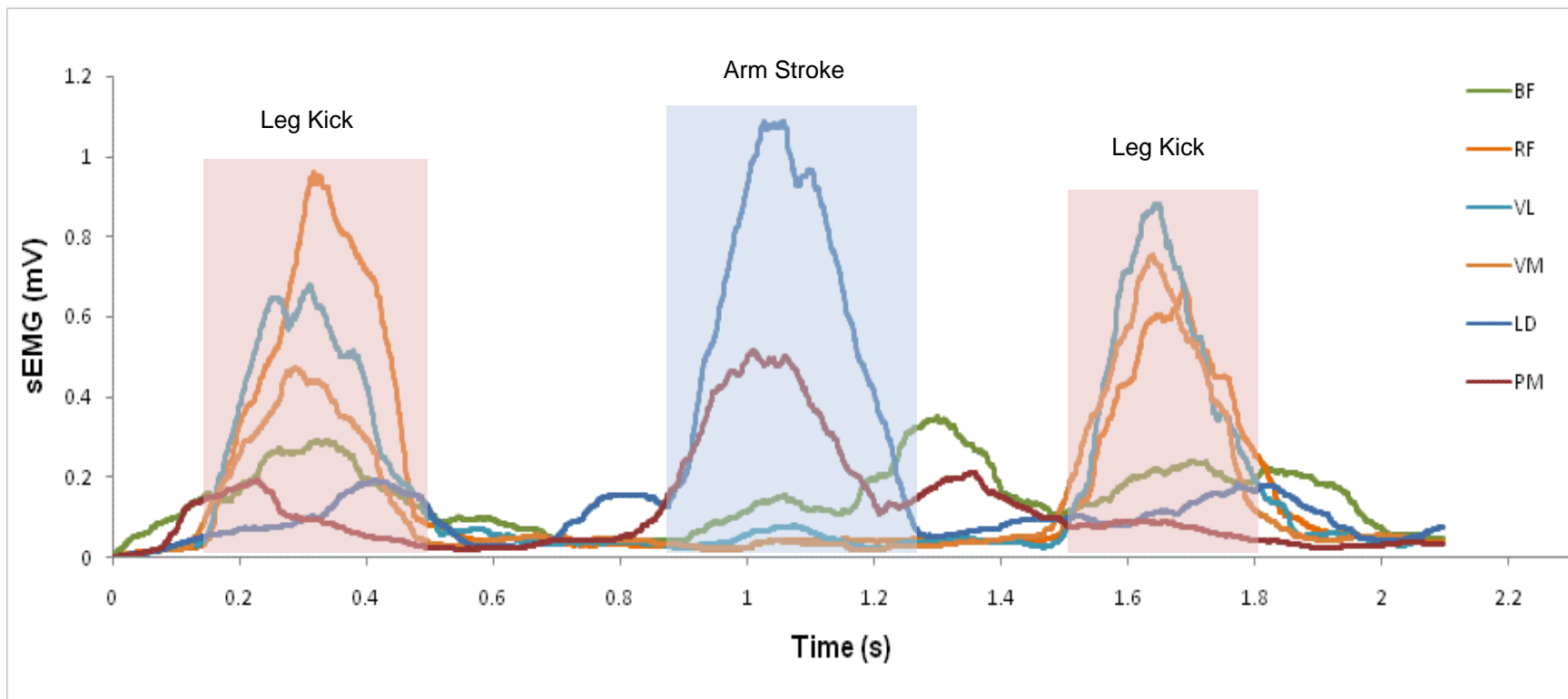
**Table 4.2** Mean ( $\pm$  SD) values of co-ordination phase one (CPhase 1) over the 1<sup>st</sup> and 2<sup>nd</sup> length of the 100 m swim.

Participant	CPhase 1 (s)	CPhase 1 (s)
	1 <sup>st</sup> 50 m	2 <sup>nd</sup> 50 m
1	0.13	0.17
2	0.03	-0.05
3	-0.06	-0.12
4	-0.03	-0.17
5	-0.19	-0.15
6	0.09	0.01
7	0.04	0.12
Mean $\pm$ SD	0.00 $\pm$ 0.11	-0.04 $\pm$ 0.12

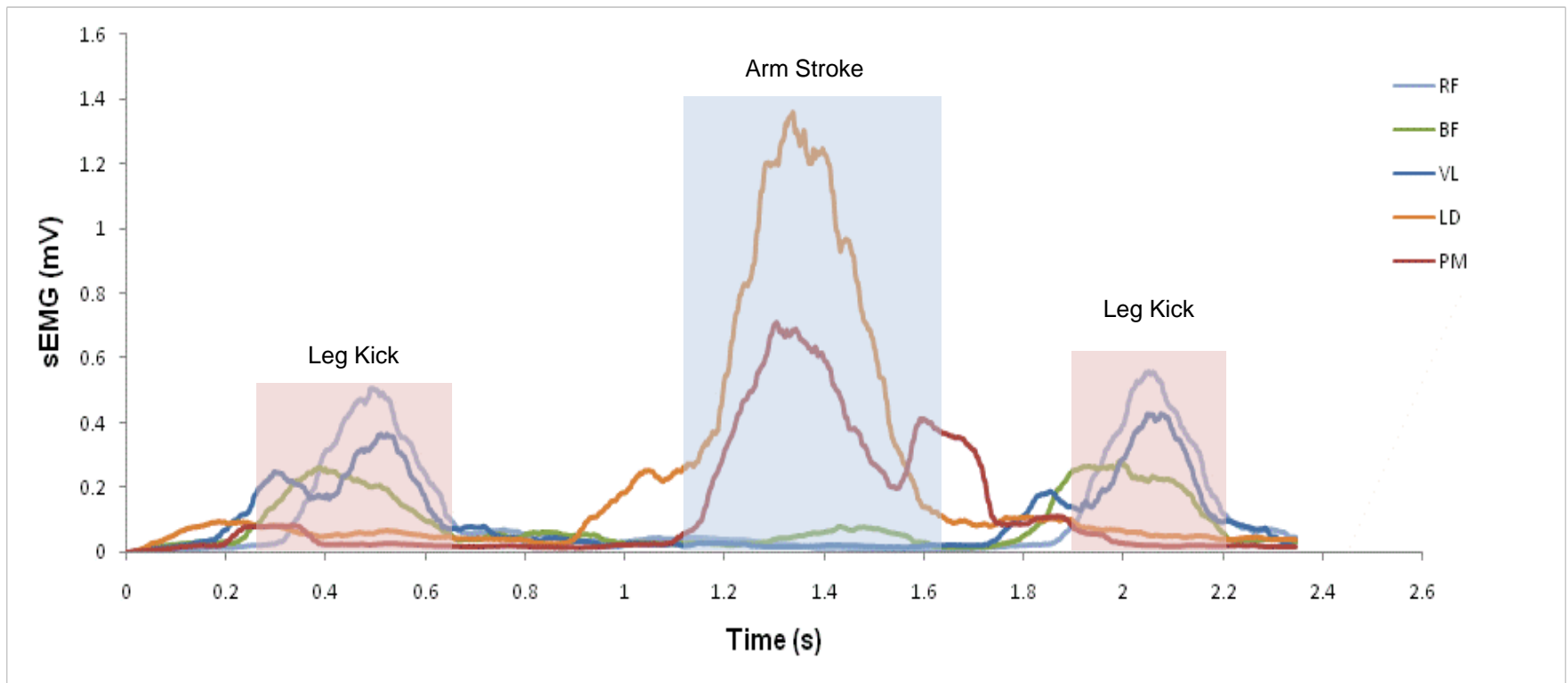
The co-ordination phase one (CPhase1) data (Table 4.2) showed that four of the seven participants utilised the glide co-ordination pattern on the 1<sup>st</sup> 50 m and the remaining three adopted the overlap co-ordination pattern. From the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m four of the participants showed an increase in CPhase 1, i.e. a shift towards the overlap co-ordination pattern (Table 4.2). Overall there was no statistically significant change in the co-ordination phase from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m  $t(6) = 1.77, p = 0.128, r = 0.56$ .



The mean muscle activation patterns of the glide co-ordination pattern and that of the overlap co-ordination (Table 4.2) were compared. There was a 23 % difference in the time between the start of the active phase of the latissimus dorsi muscle following end of the active phase of the rectus femoris muscle in the glide co-ordination technique compared to the overlap co-ordination technique. There was a similar difference of 28 % between the active phase of the pectoralis major following the end of the active phase of the rectus femoris. In the glide co-ordination technique the time from the end of the active phase of the rectus femoris to the start of the active phase of the pectoralis major was  $0.64 \pm 0.07$  s compared to  $0.65 \pm 0.08$  s to the start of the active phase of the latissimus dorsi (Figure 4.5). In the overlap co-ordination technique (Figure 4.4) the time from the end of the active phase of the rectus femoris to the start of the active phase of the pectoralis major was  $0.46 \pm 0.03$  s and  $0.50 \pm 0.05$  s to the start of the active phase of the latissimus dorsi. In the glide co-ordination technique there was a non-active phase of the rectus femoris of  $1.42 \pm 0.03$  s compared to  $1.17 \pm 0.02$  s in the overlap co-ordination technique a difference of 18 %.



**Figure 4.4** Muscle activity of the legs and arms during a single stroke cycle for participant 3 that used the overlap co-ordination technique for a single participant. Data represent the energy envelopes of muscle activity during a complete stroke cycle during the 1<sup>st</sup> 25 m



**Figure 4.5** Muscle activity of the legs and arms for a single stroke cycle for participant 1 that used the glide co-ordination technique. Data represent the energy envelopes of muscle activity during a complete stroke cycle during the 1<sup>st</sup> 25 m.

#### 4.4.3 Duration

Analysis of variance showed a significant main effect for muscle on the cycle time  $F(3,27,68.8) = 4.16, p = .007, \eta^2 = 0.22$ . Bonferoni pairwise comparisons did not identify any significant differences. There was no significant main effect for section on cycle time  $F(3,21) = 2.16, p = 0.123, \eta^2 = .236$ . There was a significant interaction between muscle and section for cycle time  $F(9,81,68.8) = 2.01, p = 0.046, \eta^2 = 0.22$ . This indicates that muscles were affected differently as they progressed through the 100 m swim.

**Table 4.3** Mean ( $\pm$  SD) and coefficient of variation percentage (CV %) for the cycle time (CT), active phase as percentage of cycle time (AF%), non-active phase as a percentage of cycle time (NAF%), across each of the four 25m sections for each of the six muscles; *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis*; *PM*, *pectoralis major*; *LD*, *latissimus dorsi*.

	1st 25 m	2nd 25 m	3rd 25 m	4th 25 m
BF CT (s)	1.27 $\pm$ 0.16 (cv=13)	1.39 $\pm$ 0.19 (cv=14)	1.44 $\pm$ 0.19 (cv=13)	1.38 $\pm$ 0.17 (cv=13)
BF AF%	40 $\pm$ 20 (cv=51)	32 $\pm$ 7 (cv=22)	27 $\pm$ 4 (cv=14)	31 $\pm$ 14 (cv=45)
BF NAF%	60 $\pm$ 20 (cv=34)	68 $\pm$ 7 (cv=10)	73 $\pm$ 4 (cv=5)	69 $\pm$ 14 (cv=20)
RF CT (s)	1.40 $\pm$ 0.14 (cv=10)	1.48 $\pm$ 0.14 (cv=10)	1.49 $\pm$ 0.17 (cv=11)	1.50 $\pm$ 0.14 (cv=9)
RF AF% <sup>#</sup>	30 $\pm$ 8 (cv=25)	25 $\pm$ 5 (cv=20)	28 $\pm$ 7 (cv=24)	27 $\pm$ 6 (cv=22)
RF NAF% <sup>#</sup>	70 $\pm$ 8 (cv=11)	75 $\pm$ 5 (cv=7)	72 $\pm$ 7 (cv=9)	73 $\pm$ 6 (cv=8)
VL CT (s)	1.12 $\pm$ 0.45 (cv=41)	1.08 $\pm$ 0.48 (cv=44)	1.50 $\pm$ 0.15 (cv=10)	1.50 $\pm$ 0.15 (cv=10)
VL AF%	41 $\pm$ 13 (cv=33)	32 $\pm$ 12 (cv=36)	32 $\pm$ 7 (cv=23)	29 $\pm$ 9 (cv=30)
VL NAF%	59 $\pm$ 13 (cv=22)	68 $\pm$ 12 (cv=17)	68 $\pm$ 7 (cv=11)	71 $\pm$ 9 (cv=12)
VM CT (s)	1.42 $\pm$ 0.12 (cv=9)	1.42 $\pm$ 0.19 (cv=13)	1.47 $\pm$ 0.14 (cv=10)	1.57 $\pm$ 0.24 (cv=16)
VM AF%	32 $\pm$ 9 (cv=27)	27 $\pm$ 5 (cv=20)	32 $\pm$ 9 (cv=27)	25 $\pm$ 10 (cv=39)
VM NAF%	68 $\pm$ 9 (cv=13)	73 $\pm$ 5 (cv=7)	68 $\pm$ 9 (cv=13)	75 $\pm$ 10 (cv=13)
PM CT (s) PM	1.45 $\pm$ 0.22(cv=15)	1.43 $\pm$ 0.15 (cv=11)	1.48 $\pm$ 0.13 (cv=9)	1.49 $\pm$ 0.19 (cv=13)
AF% <sup>#, *</sup>	44 $\pm$ 8 (cv=19)	35 $\pm$ 4 (cv=12)	33 $\pm$ 8 (cv=25)	29 $\pm$ 7 (cv=24)
PM NAF% <sup>#, *</sup>	56 $\pm$ 8 (cv=15)	65 $\pm$ 4 (cv=7)	67 $\pm$ 8 (cv=13)	71 $\pm$ 7 (cv=10)
LD CT (s)	1.18 $\pm$ 0.39 (cv=33)	1.34 $\pm$ 0.16 (cv=12)	1.62 $\pm$ 0.13(cv=8)	1.44 $\pm$ 0.10 (cv=7)
LD AF% <sup>*</sup>	32 $\pm$ 11 (cv=35)	31 $\pm$ 6 (cv=21)	21 $\pm$ 3 (cv=16)	27 $\pm$ 6 (cv=23)
LD NAF% <sup>*</sup>	68 $\pm$ 11 (cv=16)	69 $\pm$ 6 (cv=9)	79 $\pm$ 3 (cv=4)	73 $\pm$ 6 (cv=8)

<sup>#</sup> Denotes a statistically significant difference between the active phase and non-active phase of the muscle RF and PM.

<sup>\*</sup> Denotes a statistically significant difference between the active phase and non-active phase of the muscle PM and LD.

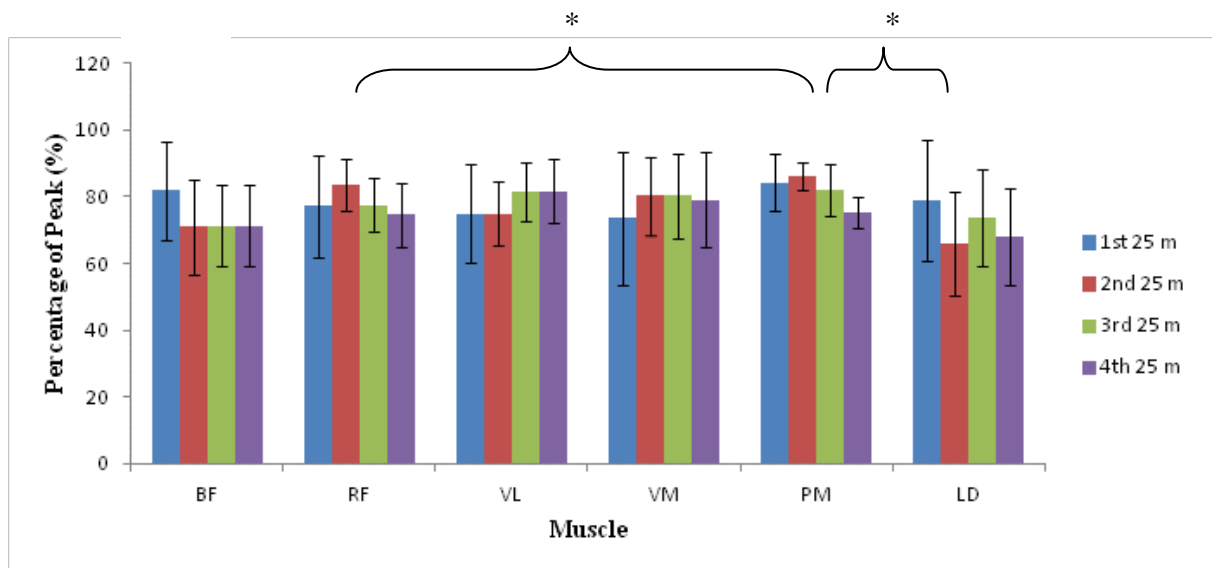
There was no significant interaction between active phase, as a percentage of cycle time, and 25 m section  $F(9.50,66.52) = 0.68, p = .729, \eta p^2 0.09$ . The relative phase of the active phase, as a percentage of cycle time, showed that there was a main effect for muscle  $F(3.17,66.5) = 3.47, p = .019, \eta p^2 0.14$  (Table 4.3). Pairwise comparisons indicated a significantly shorter (27 % shorter; Bonferroni  $p = .031$ ) active phase of rectus femoris compared to the pectoralis major. The active phase of the latissimus dorsi muscle was 28 % shorter than the pectoralis major (Bonferroni  $p = .001$ ). There was a main effect of section on active phase, as a percentage of cycle time,  $F(3,21) = 1616, p = .002, \eta p^2 0.50$ . A one-way analysis of variance showed that the active phase of the pectoralis muscle changed with 25 m section  $F(3,21) = 5.12, p = .008, \eta p^2 0.42$ . Post hoc analysis showed that the active phase of the pectoralis had significantly decreased (Bonferroni,  $p = .006$ ) by 34 % from the 1<sup>st</sup> to the 4<sup>th</sup> 25 m section of the swim (Table 4.3). There was no significant ( $p > 0.05$ ) change in the active phase of any of the other muscles over the 25 m sections of the swim, however the latissimus dorsi muscle did approach statistical significance  $F(3,28) = 2.92, p = .055, \eta p^2 0.27$ .

There was no significant interaction between 25 m section and muscle for the non-active phase when expressed as a percentage of cycle time  $F(9.50,66.52) = 94.15, p = .729, \eta p^2 0.01$  (Table 4.3). There was a significant main effect for muscle  $F(3.17,66.52) = 3.472, p = .009, \eta p^2 0.14$ . Pairwise comparisons indicated that there was a significantly longer (11 % longer;  $p = .031$ ) non-active phase of rectus femoris compared to the pectoralis major. The non-active phase of the latissimus dorsi was 12 % ( $p = .001$ ) longer than that of the pectoralis major (Table 4.3). There was a significant main effect of 25 m section on non-active phase duration, when expressed as a percentage of cycle time,  $F(3,21) = 7.11, p = .002, \eta p^2 0.50$ .

Pairwise comparisons showed that the 1<sup>st</sup> 25 m section was significantly ( $p < 0.05$ ) different to the 2<sup>nd</sup>, 3<sup>rd</sup> and 4<sup>th</sup> 25 m section. A one-way analysis of variance indicated that the non-active phase, expressed as a percentage of cycle time, of pectoralis major showed significant change over the four 25 m sections  $F(3,21) = 5.12$ ,  $p = .008$ ,  $\eta^2 0.42$ . Post hoc analysis showed that there was a 27 % ( $p = .006$ ) increase in the non-active phase of pectoralis major from the 1<sup>st</sup> to 4<sup>th</sup> 25 m section. The remaining muscles showed no significant change ( $p > 0.05$ ) in the duration of the non-active phase of muscle activity.

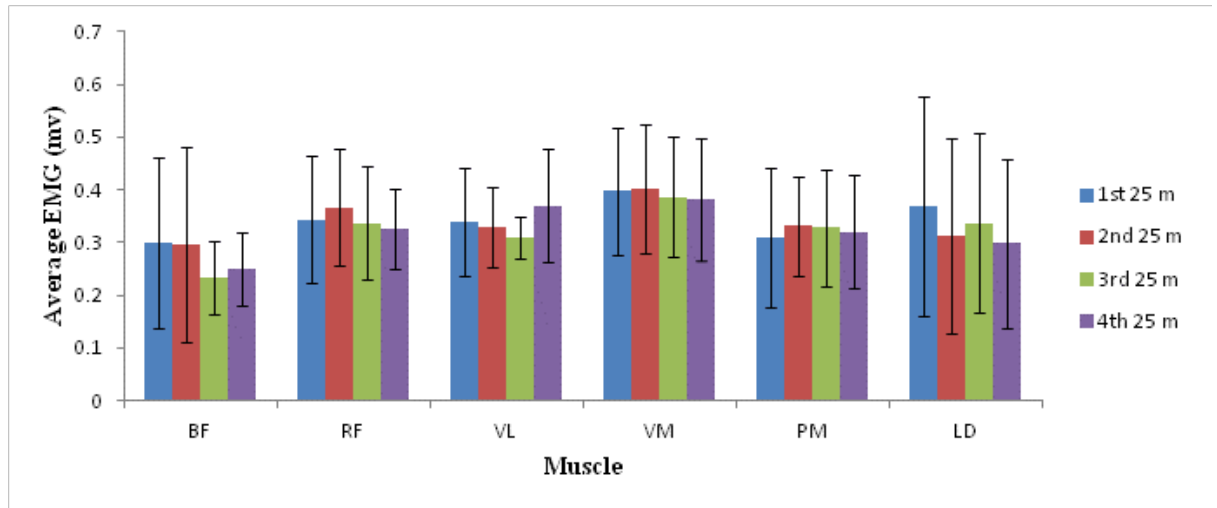
#### 4.4.3 EMG Magnitude

Two-way analysis of variance of peak sEMG as percentage of  $\text{Peak}_{Task}$  showed no significant interaction between muscle and 25 m section  $F(9.5,94.2) = 0.68$ ,  $p = .729$ ,  $\eta^2 0.09$  (Figure 4.6). There was a significant main effect of muscle on  $\text{Peak}_{Task}$ ,  $F(3.17,66.52) = 3.47$ ,  $p = .019$ ,  $\eta^2 0.14$ . Pairwise comparisons showed that there was a significantly lower (5 %;  $p = .031$ ) peak sEMG in the rectus femoris compared to the pectoralis major. There was also a significantly lower (24 %;  $p = .001$ ) peak sEMG in the latissimus dorsi when compared to pectoralis major (Figure 4.6) There was a significant main effect for section on peak sEMG,  $F(3,21) = 7.11$ ,  $p = .002$ ,  $\eta^2 0.50$ . One-way analysis of variance indicated that the peak sEMG of the pectoralis major significantly changed with lap  $F(3,21) = 5.12$ ,  $p = .008$ ,  $\eta^2 0.42$ . Post hoc analysis showed that there was a significant decrease in pectoralis major of 11 % ( $p = .007$ ) from the 1<sup>st</sup> to the 4<sup>th</sup> 25 m section of the swim. None of the other muscles showed any significant change in peak sEMG with section ( $p < 0.05$ ).



**Figure 4.6** Comparison of the normalised peak sEMG of each muscle over the four 25 m sections: *BF*, biceps femoris; *RF*, rectus femoris; *VL*, vastus lateralis; *VM*, vastus medialis; *PM*, pectoralis major; *LD*, latissimus dorsi. Note the statistical significant differences between the muscle RF and PM, PM and LD (\*  $p < 0.05$ ). Data represent mean  $\pm$  SD





**Figure 4.7** Comparison of the mean active sEMG of each muscle over the four 25 m sections: *BF*, biceps femoris; *RF*, rectus femoris; *VL*, vastus lateralis; *VM*, vastus medialis; *PM*, pectoralis major; *LD*, latissimus dorsi.

There was no significant interaction  $F(3.28, 68.85) = 1.76, p = .128, \eta^2 0.08$  between 25 m section and muscle for mean active sEMG amplitude (Figure 4.7). There was no main effect of muscle on mean active sEMG  $F(9.84, 68.85) = 4402, p = .158, \eta^2 0.02$ . There was no main effects of 25 m section on mean active sEMG  $F(3, 21) = 1.01, p = .407, \eta^2 0.13$ .

## 4.5 Discussion

### 4.5.1 Kinematics

Swimmers have been shown to adopt specific combinations of stroke length (SL) and stroke rate (SR) that change over race distance, presumably as a consequence of fatigue (Thompson, Haljand and MacLaren, 2000). This was shown in the current study where the variability in the combinations of the SL and SR (Table 4.1) indicates high levels of inter subject variability. The participant's combination of their SL and SR does not remain constant from the 1<sup>st</sup> to 2<sup>nd</sup> 50 m as shown by the decrease in SL from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. In the current study there was a 7.9% ( $p = .032$ ) decrease in SL which is similar to the 8 % decrease reported by Thompson, et al., (2000). This finding thus supports hypothesis 3. A decrease in SL has been reported to be the first sign of fatigue in swimmers (Dekerle et al., 2005; Keskinen and Komi, 1993; Weiss et al., 1988). This suggests that each individual responds differently to try and maintain clean swim speed as they progress through a swim and may differ in their rate of fatigue.

As a consequence of a decrease in SL, swimmers have to try and increase SR in an attempt to maintain clean swim speed (Thompson, Haljand and MacLaren, 2000). This is not confirmed by the findings of the current study. There was no significant change in SR from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m of the swim (Table 4.1), resulting in an overall decrease of 6.4% in the mean clean swim speed from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m (Table 4.1), which is in line with the previously reported decrease of 6 -7 % (Thompson, Haljand and MacLaren, 2000). As there was no change in SR there may have been changes in co-ordination in an attempt to offset the decrease in SL. This is in contract to that of Seifert, et al., (2009) who reported that front crawl swimmers change speed through alterations in SR and not alterations in their co-ordination. Overall there was no significant change in co-ordination phase 1 (Table 4.2) from

the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. This may be due to the high levels of inter-subject variability of in the co-ordination of the propulsive phases of the arms and legs. The co-ordination phase 1 time was characterised as the time between the end of the leg propulsion phase and the start of the arm propulsion phase (Figure 2.1, Section 2.3.4, Chapter 2). Four of the seven participants started with the glide overlap on the first lap and the remainder of the participants utilised the glide co-ordination technique. These findings are in line with those in chapter 2 (Oxford et al., 2010) that the majority of participants showed that their co-ordination pattern changed in the same direction to that of overlap or greater overlap with in the stroke cycle.

The individual alterations in the participants' co-ordination patterns in the present study could be a direct result of fatigue as shown in chapter 1 and chapter 2. Fatigue has been defined as a failure to maintain power output (Edwards, 1983) which is indicated in the current study by the decrease in clean swim speed, as was also shown in chapters 2 and 3. Fatigue has also been previously shown to hamper the sensorimotor system (Forestier and Nougier, 1998; Tripp, Yochem and Timothy, 2007) which affects the ability to maintain form and stability in movement patterns. It could be speculated that the alterations in the co-ordination patterns of the participants are a product of changes in the neuromuscular system. It has been reported that fatigue causes changes in the function of muscles that normally perform the task resulting in changes in co-ordination of muscle activity (Forestier and Nougier, 1998). These changes in muscle co-ordination and activity could result in a decreased power output which results in a decrease in clean swim speed, as shown in the current study.

#### *4.5.2 Comparison of the Muscle Activity in the Glide and Overlap Co-ordination*

##### *Techniques*

In the current study the individual changes in co-ordination of the arms and legs (Table 4.2) showed that four participants utilised the glide co-ordination and three used the overlap on the 1<sup>st</sup> 50 m. When the sEMG magnitude and amplitude is compared between the overlap and glide co-ordination techniques (Figure 4.4 and 4.5) the results show that there is a delay in the activation of the muscles of the upper body in the glide technique compared to the overlap technique. This is consistent with previous research (Leblanc et al., 2007; Chollet et al., 2004; Oxford et al., 2010; Seifert and Chollet, 2005) that has characterised: 1) the glide technique as having a glide phase following the end of the arm recovery phase and the initiation of the leg kick; 2) the overlap technique as having the initiation of the leg kick prior to the completion of the arm recovery phase.

#### *4.5.3 Comparison of the Active and Non-Active Phases of Muscle Activity*

The results of the current study show that in breaststroke swimming there are two distinct phases of muscle activity within a stroke cycle; the 1<sup>st</sup> phase of increased magnitude of the sEMG is seen in the muscles of the lower-limb and the 2<sup>nd</sup> phase of increased sEMG magnitude was seen in the upper body muscles. The 1<sup>st</sup> phase coincides with the leg kick of the stroke and the 2<sup>nd</sup> phase coincides with the arm pull phase of the stroke cycle. The leg kick and the arm pull are both propulsive phases of the stroke (Capitao et al., 2006; Yoshimurs et al., 2005). These findings are in support of previous studies (Janshen, Mattes and Tidow, 2009; Wilson, Robertson and Stothard, 1988) that have shown the importance of the vastus lateralis, vastus medialis and rectus femoris muscles in leg extension and the

importance of the muscles latissimus dorsi and pectoralis major in propulsion in swimming (Nuber et al., 1986). The magnitude of the sEMG has been shown to be proportional to the amount of force that is produced by a muscle or group of muscles (Barbosa et al., 2011).

It has been previously reported (Strin et al., 2011) that it may be better to look at the active and non-active phase as relative phases of cycle time (CT; calculated as the sum of the mean active phase and the mean non-active phase over three cycles of muscle activity). When the results are presented as relative phases of cycle time it shows that there is a statistically significant decrease in relative time of the active phase ( $p = .002$ , Table 4.3) and subsequent statistically significant increase in relative time spent in the non-active phase ( $p = .002$ ) over the four 25 m sections of the swim. The decrease in the active phase of the stroke is in accordance with previous research (Strin et al., 2011) that reported a statistically significant increase in the non-active phase and a decrease in the active phase in front crawl swimming. Although in the Strin, et al., (2011) study there was also a significant increase in the active phase of the stroke cycle for the lower part of the latissimus dorsi muscle, the upper and lower heads of the latissimus dorsi muscle were not studied as separate muscle in the current study, so a comparison is not possible. The increase in the relative time in the active phase (Strin et al., 2011) was attributed to an increase in the time spent in the propulsive phase of the stroke as shown by Seifert, et al., (2007). In the current study only the pectoralis major showed a decrease in the active phase from the 1<sup>st</sup> to the 4<sup>th</sup> 25 m section (Table 4.3). These differences between the present study and Strin et al. 2012 could be due to the different muscle activation patterns during the arm propulsion phase of the stroke of the breaststroke compared to the front crawl stroke.

The non-active phase of the stroke cycle showed no change over the duration of the 100 m swim which is not in line with the findings of Strin, et al., (2011) who reported an

increase in the relative non-active phases of the muscle during a 100 m front crawl swim. The increase in the relative time spent in the non active phase indicates that as there is an accumulation of fatigue during the swim (Strin et al., 2011) resulting in the muscle requiring longer to recover from the preceding active phase. In the current study the only muscle that showed an increase in the non-active phase was the pectoralis major muscle. The differences between the changes in relative time spent in the non-active phase of the stroke of the current study and that of Strin, et al., (2011) could be due to the natural glide and therefore recovery phases of the arms and legs which are an inherent feature of the breaststroke technique. These phases in breaststroke could be sufficient to allow the muscles time to recover thus maintaining their level of activity throughout the swim. Although the work that previous authors (Alberty et al., 2008; Seifert, Chollet and Chatard, 2007; Seifert, Chollet and Bardy, 2004; Seifert and Chollet, 2009) have conducted is not directly comparable to the current study the common findings can be outlined. It has been reported (Seifert, Chollet and Chatard, 2007) in front crawl swimmers that the relative duration of the propulsive phase was higher during the first lap compared to the final lap during a 100 m swim. These results may suggest that for the muscles to complete their task (propulsion) they need to maintain an absolute duration of activation (Strin et al., 2011). The findings of Strin, et al., (2011) also show that the muscles have to increase the relative duration of the non-active phase thus increasing the recovery time of the muscles as a result of fatigue accumulation in the muscles. However this finding is not supported by the results of the current study as the absolute value of active phase did not remain the same. These differences reported between the current study and Strin, et al., (2011) could have been due to the different strokes and the different need for propulsion in the strokes.

#### 4.5.4 *Peak EMG*

The amplitude of the muscle activity was normalised to the active peak ( $\text{Peak}_{\text{Task}}$ ) in accordance with previous research (Ball and Scurr, 2010; Burden, 2010; Clarys et al., 2010) to allow comparison between muscles. There was a significant difference between the rectus femoris muscle and pectoralis major, however the more interesting difference lies between that of the pectoralis major and the latissimus dorsi muscles. The pectoralis major muscle has been reported to have high levels of activity throughout the arm pull phase of the stroke, with the highest activity levels reported during the pull phase of the arm stroke in breaststroke swimming (Nuber et al., 1986). Previous studies in front crawl swimming have also reported that the latissimus dorsi muscle is the main swimming muscle used for the development of propulsion in the arms (Nuber et al., 1986; Strin et al., 2011). The pectoralis major and latissimus dorsi muscles are both mono-articular muscles which are used in the production of power (Wakeling, Blake and Chan, 2010). If there is a decrease in the force producing capacity of these muscles this would help explain the decrease in swimming speed that has been previously reported (Oxford et al., 2010; Takagi et al., 2004; Thompson, Haljand and MacLaren, 2000) in breaststroke swimming. As the changes in the active phase are not equal between muscles this could alter the co-activation between muscles resulting in changes in the ability of the muscles to maintain force production. At the start of the swim the co-activation of the muscles is able to produce optimal force and in the correct direction (Wakling, 2009) to optimise propulsion. As the participants progress through the swim alteration in muscle co-activation resulting in less efficient movements reduced the propulsion generated thus resulting in the decreased swim speed over a from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. The fact that there were no changes in the active phase of the latissimus dorsi muscle may mean that the latissimus dorsi is recruited at a lower level than pectoralis major thus maintaining its level of activity throughout the swim. The latissimus dorsi muscle is

known to be the main propulsive muscle in swimming (Nuber et al., 1986; Strin et al., 2011) and this may mean that there is greater reliance on the latissimus dorsi rather than the pectoralis major muscle for the maintenance of propulsion from the arm stroke in breaststroke swimming. This implies that in spite of the participants being asked to swim with maximal perceived effort the muscles that were under observation were engaged at a sub maximal level at the beginning of the swim (Strin et al., 2011). This is supported by past research that has reported that during front crawl swimming the amount of force that swimmers produce with their arms ranges between 53 and 110 N and is directly linked to swimming velocity (Hollander, De Groot and Van Ingen Schenau, 1988; Van der Vaart et al., 1987). The fact that the other muscles showed no change in the peak amplitude of the muscle activity suggests that the muscles were working at a sub maximal level at the start of the swim and that this was maintained throughout the swim. This finding is supported by previous research that has shown that muscle activity in front crawl swimming was at a sub maximal level (Strin et al., 2011; Rouard and Clarys, 1995; Wakayoshi et al., 1994).

The mean active sEMG showed no significant change over the duration of the swim (Figure 4.9) for any of the muscles studied. If the force of a muscle stays the same as can be inferred from the amplitude remaining similar over the four 25 m sections (Figure 4.8 and 4.9) there is likely to be a subsequent downward shift in the frequency spectrum of the signal (De Luca, 1979). This concept requires further investigation and cannot be commented on in the current study due to the methodological procedures using in the current study. To assess the downward shift in frequency spectrum the EMG would need to be analysed in the frequency domain (Gerdle et al., 2000; Komi and Tesch, 1979; Strin et al., 2011). A power spectral density (PSD) estimate is usually calculated from the raw EMG signal. For both



muscles there are large degrees of variability shown as indicated with the large standard deviations (Figure 4.6 and 4.7). The variability in the results could be attributed to inter and intra individual differences in muscle fibre type distributions within the muscle as well as prior mode, intensity and duration of training, which induces specific physiological adaptations to take place within the exercised muscle and other physiological systems which all relate to likely difference in fatigability characteristics of athletes (Bosco et al., 1994; Lepers et al., 2000). Other differences the current finding and previous studies could be attributed to the fact that sEMG is a non-stationary signal affected by changes in skin and tissue properties, electrode placement as well as strength of muscle contraction (Clarys and Rouard, 2010).

The rectus femoris and biceps femoris muscle peak sEMGs both showed no change from the 1<sup>st</sup> to the 4<sup>th</sup> 25 m sections due to high inter subject variability. These biarticular muscles are reported (Wakeling, Blake and Chan, 2010) to be responsible for the control of the muscle and responsible for the direction of force. Alterations in the in peak sEMGs may indicate a change in the effective application of force and go some way to explain why there is a decrease in clean swim speed over the duration of a breaststroke swim (Thompson, Haljand and MacLaren, 2000; Takagi et al., 2004; Oxford et al., 2010). As a compensatory mechanism to a change in the biarticular muscle there maybe subsequent increases in the muscle activity of the mono-articular muscle vastus lateralis and vastus medialis, which are the power producing muscles. However this needs to be further investigated with synchronised video analysis and intra-velocity fluctuations to further quantify this mechanism.

The pectoralis major muscle has been reported to have high levels of activity throughout the arm pull phase of the stroke, with the highest activity levels reported during the pull phase of the arm stroke (Nuber et al., 1986). Previous studies have also reported that the latissimus dorsi muscle is the main swimming muscle used for the development of propulsion in the arms (Nuber et al., 1986; Strin et al., 2011). The pectoralis major muscle showed a 24 % decrease in the duration of the active phase from the 1<sup>st</sup> to the 4<sup>th</sup> 25 m section compared to no change in the latissimus dorsi muscle. Both of these muscles play a key role during the in insweep of the stroke. The pectoralis major and latissimus dorsi muscles are both mono-articular muscles which are used in the production of power (Wakeling, Blake and Chan, 2010). If there is a decrease in the force producing capacity of these muscles this would help explain the decrease in swimming speed that has been previously reported (Oxford et al., 2010; Takagi et al., 2004; Thompson, Haljand and MacLaren, 2000) in breaststroke swimming. As the change in the active phase is not equal then this could decrease the co-contraction of the muscles in the production of force as both muscles would start off contracting to produce optimal force in the correct direction (Wakling, 2009) and over the duration of the swim this effectiveness may be reduced because of a lack of control in the application of force and result in a decrease in clean swim speed over a duration of a swim. The fact that there was no change in the latissimus dorsi muscle may also suggest compensatory mechanisms are being used to try and maintain force production in an attempt to maintain clean swim speed. The latissimus dorsi muscle is known to be the main propulsive muscle in swimming (Nuber et al., 1986; Strin et al., 2011) and this may mean that there is greater reliance on the latissimus dorsi rather than the pectoralis major muscle for the maintenance of propulsion from the arm stroke in breaststroke swimming.

The mean amplitude of the muscles activity showed no significant change in any muscle studied (Figure 4.7) over the duration of the swim. If the force of a muscle stays the same as can be inferred from the amplitude remaining similar over the four 25 m sections (Figure 4.6 and 4.7) there is likely to be a subsequent downward shift in the frequency spectrum of the signal (De Luca, 1979). This concept requires further investigation and cannot be commented on further in the current study due to the methodological procedures used. For all muscles there are large degrees of variability shown, as indicated by the large standard deviations (Figure 4.6 and 4.7).

The amplitude of the muscle activity has been reported (Barbosa et al., 2011) to be roughly proportional to the force exerted by the underlying muscle. However it is not a direct measure of muscle force as the relationship between muscle force and sEMG is not linear in dynamic movements (Barbosa et al., 2011). It has been reported that during repeated cyclical movements a decrease in peak tension of the muscle is correlated with an increase in the contraction time of the muscle fibres resulting in a decrease in firing frequency therefore resulting in an overall decrease in muscle force (Fuglevand, Macfield and Bigland-Ritchie, 1999; Neptune and Kautz, 2001).

#### *4.5.5 Limitations*

One of the limitations of the current study is that the stroke rate, stroke length and clean swim speed were not measured for each of the four 25 m sections of the swim. Such measurements would have enabled the relationship between the changes in muscle activity and the changes in swimming kinematics to be investigated more directly. Another limitation

of the current study was the fact that the EMG signal was not synchronised with the video so that the key phases could not be directly identified within a stroke cycle. Synchronisation of the EMG with video would have allowed for greater depth of analysis, enabling the investigation to relate any changes in muscle activity to the changes in swimming kinematics.

#### **4.6 Future Directions**

To better understand the changes in cycle time and how such changes relate to the kinematics of the stroke, EMG data needs to be viewed in conjunction with kinematic data to investigate changes in intra-muscular co-ordination and inter-muscular synchronisation. Further research is required to look at sEMG data in the frequency domain to gain a greater understanding of the effects of fatigue (Strin et al., 2011). There is also a need to further investigate sEMG with regards to understanding the changes in kinematics of the duration of a 100 m breaststroke swim. The current study does not take into consideration that differences in asymmetries in neurophysiology exist across dominant and non-dominant sides. Some of the asymmetries may include faster conduction velocities of the nerve impulse on the dominant side and differences in fibre type due to long term preferential use (Diederichsen et al., 2007). These asymmetries may have implications for the fatigue rate characteristics of dominant and non-dominant sides. Future work could monitor both sides of the body to see if there are imbalances in muscles from right to left to provide information that is critical for coaches and athletes alike. If there is unequal application in force from the left and right arms and legs then the swimmers' performances will not be maximised as the optimum muscle power will not be achieved. There may also be increased risk of injury due to unequal activation of the muscles which could lead to muscle imbalances around the shoulder (Weldon and Richardson, 2001) and knee joints (Grote, Lincoln and Gamble, 2004;

Keskinen, Eriksson and Komi, 1980) in particular potentially leading to overuse injuries which are common in swimmers (Weldon and Richardson, 2001).

#### 4.7 Conclusion

This study has investigated changes in the sEMG amplitude as a percentage of  $Peak_{Task}$  and the duration of the active and non-active phase expressed as a percentage of the muscle cycle time. The investigation involved the analysis of four key muscles in the legs and two key muscles of the upper body in breaststroke swimming over 4 sections of a 100 m maximal breaststroke swim. The main findings of the study were the significant differences in peak amplitude between muscles ( $p = .002$ ). The pectoralis major was the only muscle that showed a significant change in peak amplitude over the four sections of the swim with an 11 % ( $p = .007$ ) decrease from the 1<sup>st</sup> to the 4<sup>th</sup> section. The finding showed that there were significant differences ( $p < 0.05$ ) between muscles in the time spent in the active and non-active phase from the 1<sup>st</sup> to the 4<sup>th</sup> section. The investigation also investigated changes in the kinematics of the participants from the 1<sup>st</sup> to 2<sup>nd</sup> 50 m of the breaststroke swim with a significant decrease ( $p < 0.05$ ) in the SL and the clean swim speed. There were no significant changes in the co-ordination of the arm and legs from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. The results indicate that muscle activation was affected over the duration of the swim as there were changes in the relative times that the muscle spent in the active and non-active phase during the stroke cycle. The findings suggest a high level of reliance on the pectoralis major muscle during breaststroke swimming. However, whether these changes in the active phase and the non-active phase durations relate to the changes that have previously been shown in kinematics is currently unknown for the breaststroke swimming technique. Thus more detailed analysis is required to investigate the changes in muscular co-ordination and

kinematic changes in the stroke through the combined use of sEMG synchronised with underwater kinematic analysis.

## **CHAPTER 5**

### **FATIGUE RELATED CHANGES IN SURFACE ELECTROMYOGRAPHY IN 12MUSCLES DURING A 100 m LONG COURSE BREASTSTROKE SWIM**

## CHAPTER 5

### 5.0 Fatigue Related Changes in Surface Electromyography in 12 Muscles During a 100 m Long Course Breaststroke Swim

#### 5.1 Abstract

The aims of the study were: 1) to evaluate neuromuscular fatigue during 100 m maximum breaststroke swimming in selected upper body, trunk and lower limb muscles, by means of frequency parameters of sEMG, supported by kinematic data; and 2) to evaluate the changes in co-ordination of the arms and legs between the 1<sup>st</sup> and the 2<sup>nd</sup> 50 m of the 100 m swim. Surface electromyography (sEMG) was collected from the pectoralis major, latissimus dorsi, posterior deltoids, biceps brachii, triceps brachii, biceps femoris, rectus femoris, vastus lateralis, vastus medialis, gluteus maximus, erector spinae and rectus abdominals of seven experienced breaststroke swimmers (FINA points  $600 \pm 74$ ). Clean swim speed decreased by 6.4 % ( $p = 0.003$ ), stroke length decreased by 7.9 % ( $p = 0.03$ ) and stroke index decreased by 14 % ( $p = 0.01$ ) from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. There was no significant change ( $p > 0.05$ ) in time spent in each of the stroke phases there was also no significant change in co-ordination of the arms and legs from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. There were no significant differences ( $p > 0.05$  in each case) in EMG mean power frequency (MPF) and the median power frequency (MDF) when normalised between muscles or between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m. It was concluded that participants were not able to maintain clean swim speed from the 1<sup>st</sup> to the second 50 m due to a decrease in stroke efficiency. The decrease in stroke efficiency was not a result of significant changes in the frequency of the active muscles.



## 5.2 Introduction

In swimming the performance of a swimmer is determined by the capacity to produce propulsive forces at the same time as reducing the resistance to forward motion (Toussaint and Truijens, 2005). Propulsive forces are formed through three dimensional movements of the upper and lower limbs in response to unstable loads by the water (Payton and Bartlett, 1995). As in all human movements, fatigue can be defined as the failure to maintain a required force or power output (Edwards, 1983). However, the power generating capacity of muscle starts to progressively decrease from the onset of exercise, such that fatigue starts before the failure of the muscles to carry out the task (Gandevia, 2001). The decrease in a muscle's ability to effectively maintain power output could be due to central and/or peripheral changes (Clarys and Rouard, 2010). The central component of fatigue could be related to changes in the Central Nervous System (CNS) activations through alterations in the order of neurone recruitment and / or motor neuron activation (Clarys and Rouard, 2010). The peripheral component of fatigue could be related to alterations in the neuromuscular junction as well as changes in the availability of substrates, reduced blood flow or dysfunction of the sarcomere causing a reduction in muscle performance (Clarys and Rouard, 2010). As a consequence the study of fatigue is a complex phenomenon which has been examined through the use of different techniques such as physiology, surface Electromyography (sEMG) and mechanics (Clarys and Rouard, 2010), which are commonly combined to provide a more detailed examination of the effects of fatigue on performance.

The evaluation of fatigue via an EMG signal is usually achieved by analysis in the frequency domain (Gerdle et al., 2000; Komi and Tesch, 1979; Masuda et al., 1999). The power spectral density is normally calculated from the raw EMG signal, then the mean power

frequency (MPF) and the median power frequency (MDF) are calculated (Clarys and Rouard, 2010; Kamen and Gabriel, 2010). The MPF and MDF of the power spectral density have been shown to shift to lower frequencies during fatiguing exercises (De Luca, 1979; Merletti and Lo Conte, 1997). The decreases in the MPF and MDF frequencies have been attributed to declining muscle fibre conduction velocity (MFCV) as a result of local metabolic changes in the working muscle (Masuda, Miyano and Sadoyama, 1983). Other explanations for the alteration in the power spectral density of MDF and MPF include modifications in motor unit (MU) action potential shape and firing rate and the synchronisation of firing of the MUs (Brody et al., 1991; Bigland-Ritchie and Woods, 1984; Gabriel and Kamen, 2009).

During dynamic cyclic movements there are high levels of non-stationarity in the EMG signals causing the signal properties to alter rapidly with time (Strin et al., 2011). To be able to consider the spectral properties of such signals, the signals must be evaluated using procedures that use simultaneous analysis of the signal properties in the time and frequency domains (Strin et al., 2011).

There are various methods available to analyse the time – frequency domain of the EMG signal such as the Cohen-Class transformations (Bonato et al., 2001; Knaflitz and Bonato, 1999), the continuous wavelet transform (Clarys and Rouard, 2010; Karlsson, Yu and Akay, 2000). However simpler methods of short-time Fourier transforms can be applied effectively in the analysis of dynamic muscle contractions for decreases in the MPF and MDF commonly associated with the development of fatigue (Christensen et al., 1995; Gerdle et al., 2000; Mac Issac, Parker and Scott, 2001)

Only a few studies in swimming have analysed the sEMG in the frequency domain (Aujouannet et al., 2006; Caty et al., 2007; Strin et al., 2011). A study by Strin, et al., (2011) investigated the changes in the MPF of the upper and lower sections of the latissimus dorsi and pectoralis along with the biceps brachii and triceps brachii in a 100 m front crawl swim. The authors reported a linear decrease in the MPF in all the muscles studied, with the greatest decreases in MPF, of about 25 %, taking place in latissimus dorsi and triceps brachii. Strin, et al., (2011) also reported no statistically significant difference in relative decrease in MPF between the four muscles studied ( $p>0.05$ ). Other swimming studies have reported changes in the frequency domain including Aujouannet, et al., (2006) who looked at the isometric contractions of the arm extensor muscles before and after four maximal effort 50 m swims and reported a decrease in the MPF of the EMG power spectrum. The only other study that has investigated changes during a swim is the study by Caty, et al., (2007) which reported an 11 % and 9 % decrease in the MPF of the extensor carpi ulnaris and the flexor carpi ulnaris, respectively, during four 50 m swims. To better understand the fatigue process during breaststroke swimming, the role of individual muscles should be considered. All previous research that has been conducted on fatigue in swimming has been on front crawl swimming. The two strokes are distinctly different in the usage of the arms and legs due to the technique constraints, thus the findings of studies on front crawl cannot be generalised to breaststroke.

A study by Aujouannet, et al., (2006) reported a decrease in isometric force measured after four 50 m maximal effort swims in front crawl in the arm flexors. This is further supported by the work of Toussaint, et al., (2006) who has also reported a decrease in mechanical power output during a 100 m all out effort arms only front crawl swim using hand force recordings in the Measure of Active Drag (MAD system). Toussaint, et al., (2006) found that the reduced force produced by the fatiguing muscle meant that, the swimmers

were unable to maintain stroke length (SL), which has been used as one of the first indicators of fatigue in swimming (Dekerle et al., 2005; Keskinen and Komi, 1993; Weiss et al., 1988). Swimmers often compensate for a decrease in SL with an increase in stroke rate (SR) to maintain swim speed (Alberty et al., 2008), but as fatigue progresses, the SR and SL, and therefore swim velocity, decrease as well (Changalar & Brown, 1992; Kennedy, et al., 1990; Oxford, et al., 2010; Takagi, et al., 2004; Thompson, et al., 2000; Chapter 2). Stroke Index (SI) is a product of average velocity and stroke length and has been shown to be a valid indicator of stroke efficiency (Costill, et al., 1985). If the maximal force generating capacity of the muscle declines from the start of the activity due to the onset of fatigue the muscles will fail to perform the task (Gandevia, 2001) resulting in changes in stroke index (SI).

The major leg muscles that are used in the breaststroke leg kick are: vastus lateralis and vastus medialis, which play an important role in power production during extension of the knee joint (Janshen, Mattes and Tidow, 2009; Ryan and Gregor, 1992; Wilson, Robertson and Stothard, 1988); the rectus femoris that is also used for extension of the knee joint; the knee flexor and hip extensor muscle biceps femoris that is active during the end of knee extension and also plays a role in hip extension (Hug and Dorel, 2009). The important muscles of the upper body during breaststroke are: posterior deltoid, which has been shown to be the most active of the three heads of the deltoids during the whole stroke with peak activity occurring during the early pull through phase (Ruwe et al., 1994). The posterior deltoid muscle is active in the extension and outward rotation of the glenohumeral joint; the pectoralis major and latissimus dorsi that work together in the extension, adduction and internal rotation of the glenohumeral joint during the arm pull phase of the stroke cycle. The pectoralis major has been reported to produce high levels of activity throughout the arm recovery phase and at the beginning and middle of the arm pull phase (Nuber et al., 1986).

The latissimus dorsi muscle has been shown to demonstrate activity during the pull phase of the arms with peak activity at the midpoint of the arm pull phase (Nuber et al., 1986); the biceps brachii muscle has been reported to show low activity throughout the stroke (Nuber et al., 1986). The triceps brachii muscle is used to fully extend the arm during the extension of the arms (Nuber et al., 1986).

The majority of studies that have investigated sEMG changes during swimming have concentrated on the activity of the upper body (Caty et al., 2007; Pink et al., 1991; Rouard and Clarys, 1995; Strin et al., 2011; Wakayoshi et al., 1994). Only the study by Ikuta, et al., (2012) has investigated the muscle activity of the lower limb muscles and that was in front crawl swimming. None of the previous research has considered the importance of the hips and trunk in swimming. The hip constitutes the gluteals, which includes the muscles gluteus maximus, gluteus medius and gluteus minimus. The trunk is comprised of the rectus abdominals, external obliques, and transverse obliques, which are located in the front and the lateral sides of the trunk. In the back are the major spine extensors, which include the erector spinae. All these muscles function to provide postural stability (Prins, 2007). This is particularly important when the upper and lower extremities attempt to produce force during such actions as pulling, lifting and carrying (McGill, 2001; McGill, 2002). If the muscles of the arms and legs are to be able to generate sufficient propulsive force while swimming, this needs to be done from a stable base of support (Prins, 2007).

In breaststroke swimming it has been reported that during a 100 m swim (Oxford, et al., 2010; Takagi, et al., 2004; Chapter 2) there are changes in co-ordination patterns with a shift towards increased overlap in the stroke or shift towards the overlap technique. In front crawl swimming it has been reported that there is a decrease in the duration of non-propulsive

phases of the arm stroke as a result of increased fatigue (Toussaint, et al., 2006). These kinematic changes occur as the swimmer attempts to spend a greater proportion of the stroke cycle in the propulsive phase of the stroke to avoid a reduction in clean swim speed (Alberty et al., 2008; Seifert, Chollet and Chatard, 2007).

In breaststroke swimming a swimmer may adopt a unique combination of stroke rate (SR) and stroke length (SL) to achieve their optimum speed. This optimum combination will be related to the individual co-ordination pattern that is utilised by the swimmer. In competitive breaststroke swimming three distinct styles of co-ordination have been observed, *Glide*, *Continuous*, and *Overlap* (Maglischo, 2003; Oxford, et al., 2010; Chapter 1). Previous studies that have investigated changes in co-ordination in breaststroke swimming during a 100 m swim have reported a change in the co-ordination of the arm and legs with a shift towards the overlap technique by reducing the glide phase or by increasing the overlap within the stroke (Oxford et al., 2010). The changes in the co-ordination patterns previously reported (Oxford et al., 2010) suggest that these were compensatory mechanisms used by the swimmers in an attempt to maintain swim speed. It has been previously reported (Bonnard et al., 1994; Forestier and Nougier, 1998; Sparto et al., 1997) that compensatory mechanisms of fatigue involve other muscles taking over the function of the muscles that normally perform a cyclic task. To date there are no studies that have investigated changes in both muscle activity and co-ordination patterns in breaststroke swimming.

A greater understanding of the activation changes that occur within the muscles and the simultaneous changes in co-ordination patterns within the stroke would give sport scientists, coaches, and physicians a greater understanding of swimming performance. This

information could help the design of interventions (Pelayo et al., 2007) to minimise the predisposition to injury and to maximise performance during training and competitions.

### *5.2.1 Aims*

The aims of the study were: 1) to evaluate fatigue during 100 m maximum breaststroke swimming in selected upper body, trunk and lower limb muscles by means of frequency parameters of sEMG, supported by kinematic data; and 2) to evaluate the changes in co-ordination of the arms and legs between the 1<sup>st</sup> and the 2<sup>nd</sup> 50 m of the 100 m swim to determine whether such compensatory mechanisms were used in an attempt to offset the effects of fatigue during 100 m breaststroke swimming.

### *5.2.2 Hypotheses*

1) There will be a decrease in clean swim speed from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m and a simultaneous decrease in stroke length.

2) All the selected muscles will show a decrease in frequency parameters of sEMG over the duration of the swim.

3) The main power producing muscles of the upper body, pectoralis major, and of the lower body, vastus medialis and vastus lateralis, will show the largest decrease in frequency parameters of sEMG over the duration of the swim.

4) The co-ordination of the swimmers will move towards increased overlap from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m with a decrease in the time spent in the co-ordination phase one (CPhase1) of the stroke, in an attempt to compensate for the effects of muscle fatigue.

## 5.3 Methods

### 5.3.1 Participants

Participants were seven male competitive breaststroke swimmers, aged  $19.8 \pm 2.1$  years, body mass  $75.6 \pm 3.6$  kg, height  $1.81 \pm 0.60$  m. All participants were members of the ASA National Beacon Squad, FINA points  $600 \pm 74$  based on FINA points scoring 2012 for 100 m long course for breaststroke (Splash; FINA point calculator; GeoLogix, Bern, Switzerland). The swimmers' regular weekly training schedule consisted of approximately 12-20 h of swimming along with 2-4 h of dry land training. The participants were asked to refrain from heavy strength training 48 h prior to the study. The procedures and the purpose of the study were fully explained to participants and they all signed the informed consent forms. The study was approved by the Coventry University Ethics Committee.

### 5.3.2 Testing Procedure

All testing took place in the same 50 m pool at the same time of day during morning swimming between the hours of 6 and 8 a.m. For sEMG analysis the pool was split up into 25 m sections (Figure 4.2). Once the participants had prepared for EMG data collection they completed a 500 meter warm up. The warm up consisted of a series of ten 50 m swims at medium level effort (Strin et al., 2011). After the warm up the participants were instructed to complete a 100 m breaststroke maximal effort swim, with no race strategies, from a water start.



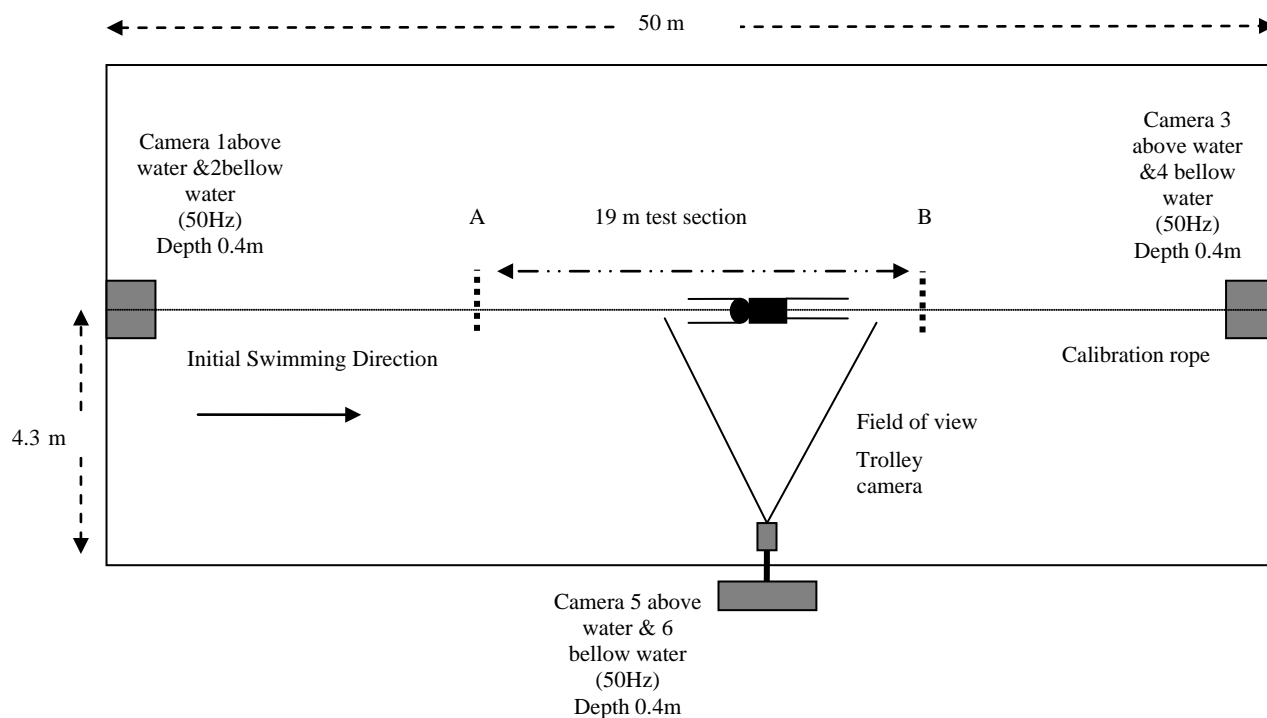


Figure 5.10 A plan view of the filming set-up used for qualitative analysis.

### 5.3.3 Swim Trials

Time to complete 100 m was recorded using a hand held stop watch (Fastime 7, AST, Leicestershire, UK) as the time from when the feet left the wall at the start until the double hand touch on the wall at the end of the swim.

Five digital video cameras (Sony video HDR-HC9, Sony, Tokyo, Japan), sampling at 50Hz with a shutter speed of 1/300 s were used to film the participants above and below the water. One camera was placed above the water at a height of 0.6 m and one camera, enclosed in a custom made waterproof housing (Plate 1.0; Chapter 2), was placed below the water at a depth of 0.4 m, at each end of the lane to record frontal and rear views (Figure 5.10). The fifth camera (Sony video HDR-HC9, Sony, Tokyo, Japan) was attached above water on the trolley. The field

of view was adjusted on all cameras so that the whole body of the participant was visible (Figure 4.1). A sixth underwater camera, a waterproof bullet camera, connected to a visual display unit (VDU; Sony digital video cassette recorder GV-D800E, Sony, Tokyo, Japan) was attached to a trolley (Figure 2.0, Chapter 2, Section 2.3.2). The frontal and rear views of the participant were synchronised to the sagittal view (Qunitic, V17, Warwick, UK) using a custom made LED light trigger system that could be seen in all six camera views. The trolley was manually moved at the same speed as the participants, parallel to the greater femoral trochanter to maintain the joint marker in the approximate centre of the field of view (Figure 4.1), in the sagittal plane, throughout the entire 100 m swim. Refraction due to the water was not accounted for in this study as refraction in water due to light rays is relatively low, with previously reported values that vary between 0.39 and 1.28 % (Gourgoulis et al., 2008). None of the digital video footage from the digital cameras were not synchronised with the sEMG signal.

#### 5.3.4 Stroke Parameters

The following stroke parameters were calculated from the 19 m testing sections of the pool on each of the two laps of a 100 m swim from the sagittal view video recordings (trolley camera, Figure 5.10). *Clean Swim speed* ( $\text{m}\cdot\text{s}^{-1}$ ) was defined as the mean forward speed of the greater trochanter over the 19 m testing section on each lap (Figure 5.10), using both above and below water camera views to monitor from the time the greater trochanter entered the 19 m testing section until it left the 19 m testing section (Figure 4.1) via the stop clock located on the video analysis software (Qunitic, V17, Warwick, UK) (to the nearest  $0.01 \text{ m}\cdot\text{s}^{-1}$ ); *Stroke frequency* ( $\text{stroke}\cdot\text{min}^{-1}$ ) was defined as the number of stroke cycles performed in one minute, calculated for each 19 m testing section (Figure 2.1) (to the nearest  $0.01 \text{ strokes}\cdot\text{min}^{-1}$ ); *Stroke length* ( $\text{m}\cdot\text{cycle}^{-1}$ ) was defined as the horizontal distance that the participants greater trochanter

travelled in one stroke cycle, computed from the clean swim speed and the stroke frequency values (to the nearest 0.01m) (Equation 2.1, Section 2.3.5); *Stroke Cycle Time* (s) was defined as the time taken to complete one complete stroke cycle, calculated as the mean stroke cycle time over each 19 m testing section (to the nearest 0.02 s); Stroke index (SI) as defined by Costill et al., (1985) (Equation 2.2; Chapter 2) was used to evaluate swimming efficiency; Co-ordination phase 1 (CPhase1) (Chapter 2) was calculated as the time between the end of the leg kick phase and the start of the arm recovery phase and was used to classify each participant's co-ordination as overlap (represented by a negative value to the nearest 0.02 s indicating simultaneous propulsion of the arms and legs), glide (represented by a positive value to the nearest 0.02 s indicating a delay (glide) in the initiation of the arm pull phase) or continuous.

### 5.3.5 *Arm and Leg Co-ordination and Stroke Phases*

Three complete stroke cycles (Chollet et al., 2004) which were completed at the end of each of the 19 m testing section (Figure 5.1) were analysed using the synchronised frontal and sagittal camera views (Figure 5.1) to determine the average duration of each of the following phases: Arm Pull (the time between the separation of the hands from the extended position in front of the body until the first forward movement of the elbow when the hands were under the head); Arm recovery (the time between the end of the arm pull phase and the start of the separation of the hands from the extended position); Leg kick (the time between the start of the first backwards movement of the feet, the point where the legs were maximally flexed at the start, and the point when the legs were fully extended); Leg glide (the time between the end of the leg kick phase and the first movement of the feet towards the buttocks); Leg recovery (the time between the end of the leg glide phase and the complete flexion of the knee until forward movement of the feet had finished); Co-ordination phase 1 (CPhase1) was calculated as the time

between the end of the leg kick phase and the start of the arm recovery phase and was used to classify the participants co-ordination as overlap (represented by a negative value to the nearest 0.02 s indicating simultaneous propulsion of the arms and legs), glide (represented by a positive value to the nearest 0.02 s indicating a delay (glide) in the initiation of the arm pull phase) or continuous; Co-ordination phase 2 (CPhase 2; the time between the end of the arm pull phase and the start of leg kick phase); Arm lag time (ALT; corresponded to the time from the start of the leg kick to the beginning of arm pull). All phases were expressed as a percentage of total cycle time with a precision of 0.02 s (Figure 2.2 Chapter 2). It should be noted that the start of the arm pull phase and the end of the arm pull phase as described above does not necessarily correspond the start and end of the propulsive components of the arms' stroke, similarly for the leg kick, the start of the leg kick and the end of the leg kick does not necessarily correspond to the start and end of the leg propulsion (Maglischo, 2003). The key stroke phases of the arms and legs were subjectively determined by three independent operators using a blind technique i.e. without knowing the results of the other operators. The three independent analyses were then compared with the mean difference of the operators being ( $< 0.04$  s to the nearest 0.02 s), which was less than the 0.04 s which has previously been used to validate key stroke phases (Seifert, Chollet and Chatard, 2007).

### 5.3.6 *Collection of the EMG Data*

Passive bipolar surface electrodes of 30 mm diameter (Blue Sensor, Ltd, Denmark, Ag/AgCl) were used to measure muscle activation of the pectoralis major, latissimus dorsi, anterior deltoid, bicep brachii, tricep brachii, rectus abdominals, erectus spinale, gluteus maximus, rectus femoris, vastus lateralis, vastus medialis and bicep femoris. The Surface electrodes were placed on the contracted belly of each of the muscles, in line with the muscle

fibre direction in accordance with recommendations of SENIAM (Surface EMG for Non-Invasive Assessment of Muscles) (Hermens and Freriks, 1997) with an inter electrode distance of 1.5 cm (Basmajian, 1973). Before electrode placement, each participant's skin was prepared in accordance with International Society of Electromyography and Kinesiology (ISEK); the area was shaved, in order to reduce skin impedance and cleaned with alcohol wipes. The electrodes were covered with a waterproof transparent dressing to prevent water damage to the electrodes (Rouard and Clarys, 1995). Vaseline was applied to the edges of the dressing to ensure waterproofing. Adhesive elastoplast tape was used to fix the cables to the skin, to avoid interference with the signal and the participant whilst swimming (Strin et al., 2011). Electromyography activity was recorded via an ME6000 system (Mega Electronics Ltd., Finland), with an input impedance of less than  $10^{15}/0.2$  ohm/pF, a common mode rejection ratio at 60 Hz of greater than 110dB, a noise level of 1.2 mV, a gain of  $10 \pm 2\%$  and a bandwidth range from 0 Hz – 500 Hz. Muscle activity was sampled at 1000 Hz via a 16bit DAQ-516 A/D card and stored on a laptop computer using MegaWin software (Mega Electronics Ltd., Finland).

The telemetric EMG device (MEGA ME6000) was encased in a custom made water proof housing and fixed to a rod. A research assistant walked along poolside, next to the participant to keep the EMG device (MEGA ME6000) and cables free of the participants. As a result of the measurement equipment, the participants started with a push start from the side of the pool.



**Plate 5.1** Shows a side view of the location of the EMG device in its waterproof housing and its location relative to the participant.

### 5.3.6 *EMG Signal Processing*

Raw EMG signals were rectified and averaged to obtain full wave signals. All signals were visually inspected during real time collection of EMG to ensure optimal signal quality. Some EMG electrodes were exposed to water due to failure of the waterproof taping, so the EMG data was contaminated with noise and discarded from the analysis. The median frequency (MDF) and mean power frequency (MPF) were calculated using a Fast Fourier Transformation (FFT) algorithm and a Hamming window function (Pitcher, Behm and MacKinnon, 2007). This was a data reduction option available from the MegaWin software (Mega Electronics Ltd, Kuopio, Finland) employed in the EMG data collection and analysis. A single spectral estimate was calculated using a 1024 point moving window, with a 50% overlap. The slope of the MDF and MPF change (DH<sub>z</sub>/s) was

calculated in the MegaWin programme for the 1<sup>st</sup> 50 m, 2<sup>nd</sup> 50 m performance respectfully, where a linear regression analysis was performed as an estimate of the rate of change over time (Strin et al., 2011). For each 50 m lap, the first and last five cycles of muscle activity (one muscle cycle was calculated from the onset and offset activity of the rectus femoris muscle) were removed from the analysis to remove the effects of start, turns and finishing techniques. To calculate the changes in activity over the whole 100 m swim, the 5 cycles of muscle activity of the rectus femoris at the beginning and end of each 50 m were disregarded to reduce the effects of the start, turns and finishing. The changes in MDF and MPF were calculated for the three time periods, corresponding to the 1<sup>st</sup> 50 m, 2<sup>nd</sup> 50 m and overall performance, and were employed as an estimate for muscular fatigue in accordance with Pitcher, et al., (2007). The values of MPF and MDF belonging to each muscle for the first (MPF<sub>Beg</sub> and MDF<sub>Beg</sub>) and last stroke (MPF<sub>End</sub> and MDF<sub>End</sub>) for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m excluding the first and last five strokes of each lap to remove the effect of starts, turns and finishing were calculated. To normalise the results between subjects and muscles, the final MDF and MPF values were expressed as a percentage of the initial values and labelled MDF<sub>n</sub> and MPF<sub>n</sub> as shown in equation 5.1 and 5.2 respectfully (Strin et al., 2011).

$$\text{Equation 5.1} \quad \text{MDF}_n = \frac{\text{MDF}_{\text{End}}}{\text{MDF}_{\text{Beg}}} \times 100$$

$$\text{Equation 5.2} \quad \text{MPF}_n = \frac{\text{MPF}_{\text{End}}}{\text{MPF}_{\text{Beg}}} \times 100$$

### 5.3.7 Statistical Procedures

Statistical analyses were conducted using SPSS version 16.0 (SPSS, Inc., Chicago, IL, USA). Standard statistical methods were used to calculate mean and standard deviation for all measured variables. Normal distribution of the data was verified using Levenes test. The percentage data was tested for normal distribution using the  $z$ -score of skewness and kurtosis in accordance with Vincent, (2005). If normal distribution was not reported then data was corrected for heterogeneity of variance using the Arcsine transformation (Black, 1999). The level of significance was set at  $p < 0.05$ . Independent T tests were used to determine differences within stroke rate (SR), stroke length (SL) clean swim speed (CSS), stroke index and the relative phases of the arms and legs from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. Independent T tests were used to determine any differences in the rate of change in MPF and MDF from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m for muscles biceps femoris ( $n = 6$ ), rectus femoris ( $n = 5$ ), vastus lateralis ( $n = 5$ ), vastus medialis ( $n = 6$ ), gluteus maximus ( $n = 5$ ), rectus abdominals ( $n = 5$ ), erectus spinale, ( $n = 6$ ), posterior deltoid ( $n = 6$ ), pectoralis major ( $n = 5$ ), latissimus dorsi ( $n = 6$ ), triceps brachii ( $n = 6$ ) and biceps brachii ( $n = 6$ ). The effect size of the independent T test was estimated using Pearson's correlation coefficient in accordance with Rosnow & Rosenthal, (2005), with values interpreted according to Cohen, (1988) as  $r = 0.10$  (small effect),  $r = 0.30$  (medium effect) and  $r = 0.50$  (large effect) results and reported in accordance with Field (2009). Analysis of Variance was used to analyse the change in  $MDF_{Beg}$ ,  $MDF_{End}$  and  $MPF_{Beg}$ ,  $MPF_{End}$  for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for each muscle. Two-way Analysis of Variance was used to analyse the change in normalised  $MDF_n$  and  $MPF_n$  with muscles ( $n = 12$ ) and lap (1<sup>st</sup> and 2<sup>nd</sup> 50 m) as the fixed factors. When Mauchley's test of sphericity indicated a minimal level of violation ( $> 0.75$ ) the degrees of freedom



was corrected using the Huynh-Feldt adjustment and when the sphericity was  $< 0.75$ , the Greenhouse-Geiser correction was used (Field, 2009). Where differences were noted in ANOVA, pairwise comparisons (Bonferroni adjusted) were employed to identify where the significant differences occurred. A level of  $p < 0.05$  was considered statistically significant. The effect size for the ANOVA statistics was estimated using partial Eta squared ( $\eta^2$ ) for analysis of variance. Since this method is likely to overestimate effect sizes, values were interpreted according to Ferguson (2009) as no effect if  $0 \leq \eta^2 < 0.05$ ; a minimum effect if  $0.05 \leq \eta^2 < 0.26$ ; a moderate effect if  $0.26 \leq \eta^2 < 0.64$ ; and a strong effect if  $\eta^2 \geq 0.64$ . All ANOVA results were reported in accordance with Field (2009).

## 5.4 Results

### 5.4.1 Performance Data

**Table 5.1** Mean ( $\pm$  SD) and the coefficient of variation as a percentage (CV %) for the values of clean swim speed, stroke length, stroke rate, and stroke index for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m swim.

	1 <sup>st</sup> 50 m	2 <sup>nd</sup> 50 m
Clean Swim Speed ( $\text{m}\cdot\text{s}^{-1}$ )	1.20 $\pm$ 0.03 (cv=3)	1.12 $\pm$ 0.04 (cv=4)*
Stroke Length ( $\text{m}\cdot\text{cycle}^{-1}$ )	1.64 $\pm$ 0.09 (cv=6)	1.51 $\pm$ 0.17 (cv=12)*
Stroke Rate ( $\text{stroke}\cdot\text{min}^{-1}$ )	44.3 $\pm$ 2.2 (cv=5)	45.3 $\pm$ 4.4 (cv=10)
Stroke Index ( $\text{m}^2\cdot\text{s}^{-1}$ )	1.97 $\pm$ 0.14 (cv=7)	1.70 $\pm$ 0.23 (cv=14)*

\*Denotes a statistically significant difference  $p < 0.05$  between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m

Overall there was a statistically significant  $t(6) = 4.74$ ,  $p = 0.003$ ,  $r = 0.89$ , decrease of 6.4 % in CSS from the 1<sup>st</sup> 50 m to the 2<sup>nd</sup> 50 m (Table 4.1). There was a statistically significant  $t(6) = 2.76$ ,  $p = .032$ ,  $r = 0.75$ , decrease of 7.9 % in SL from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. There was no change in SR,  $t(6) = -0.91$ ,  $p = 0.28$ ,  $r = 0.35$ , from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m of the swim. There was a statistically significant  $t(6) = 3.75$ ,  $p = 0.01$ ,  $r = 0.82$ , decrease of 14 % in the SI from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m of the swim.

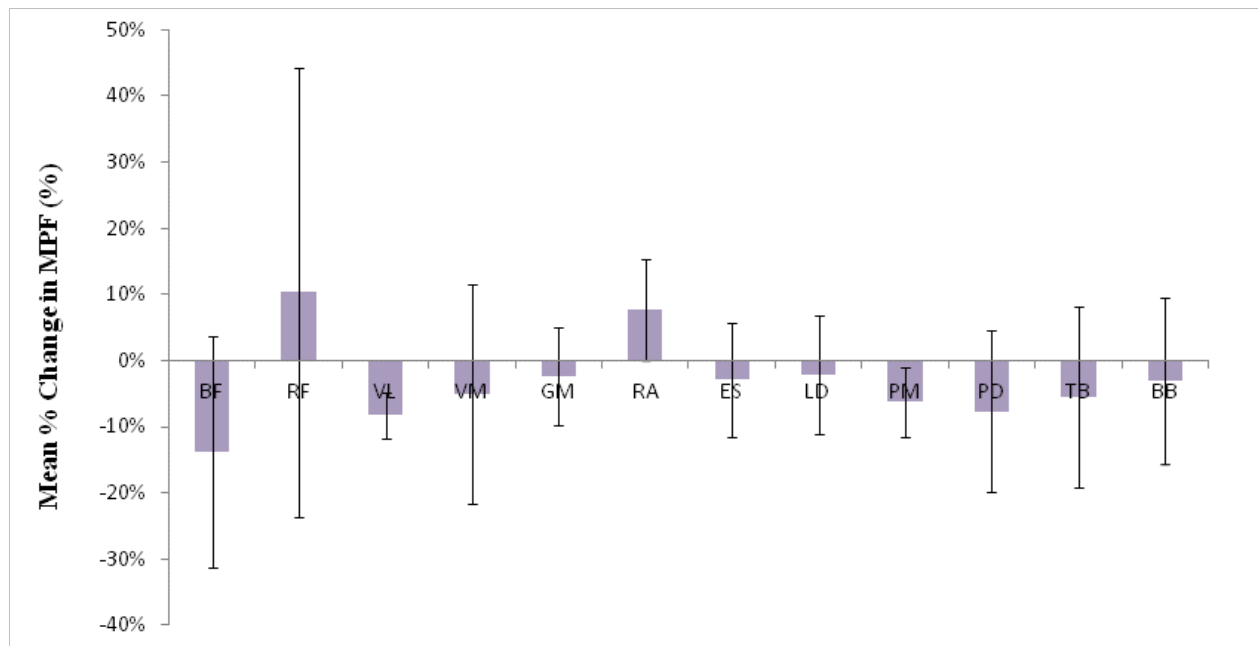
#### 5.4.2 Co-ordination Changes

**Table 5.2** Mean ( $\pm$  SD) values and the coefficient of variation as a percentage (CV %) of arm and leg stroke phases and arm-leg co-ordination expressed as a percentage over the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of the 100 m swim. ALT = Arm leg lag

	1 <sup>st</sup> 50 m	2 <sup>nd</sup> 50 m
Kick Propulsion (%)	23.4 $\pm$ 3.8 (cv=16)	23.4 $\pm$ 1.6 (cv=7)
Kick Glide (%)	47.8 $\pm$ 7.4(cv=15)	48.4 $\pm$ 4.7(cv=10)
Kick Recovery (%)	28.8 $\pm$ 5.2(cv=18)	28.3 $\pm$ 3.9(cv=14)
Arm Propulsion (%)	52.9 $\pm$ 7.1(cv=13)	54.4 $\pm$ 5.1(cv=9)
Arm Recovery (%)	47.1 $\pm$ 7.0(cv=15)	45.6 $\pm$ 5.1(cv=11)
Transition (%)	-0.2 $\pm$ 7.4(cv=3190)	-2.8 $\pm$ 7.6(cv=271)
ALT (%)	22.8 $\pm$ 5.1(cv=22)	19.4 $\pm$ 4.3(cv=23)

When the results were expressed as percentage of stroke cycle time (Table 5.3) there was no statistically significant change in the phases from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m.

### 5.4.3 Overall Changes in EMG

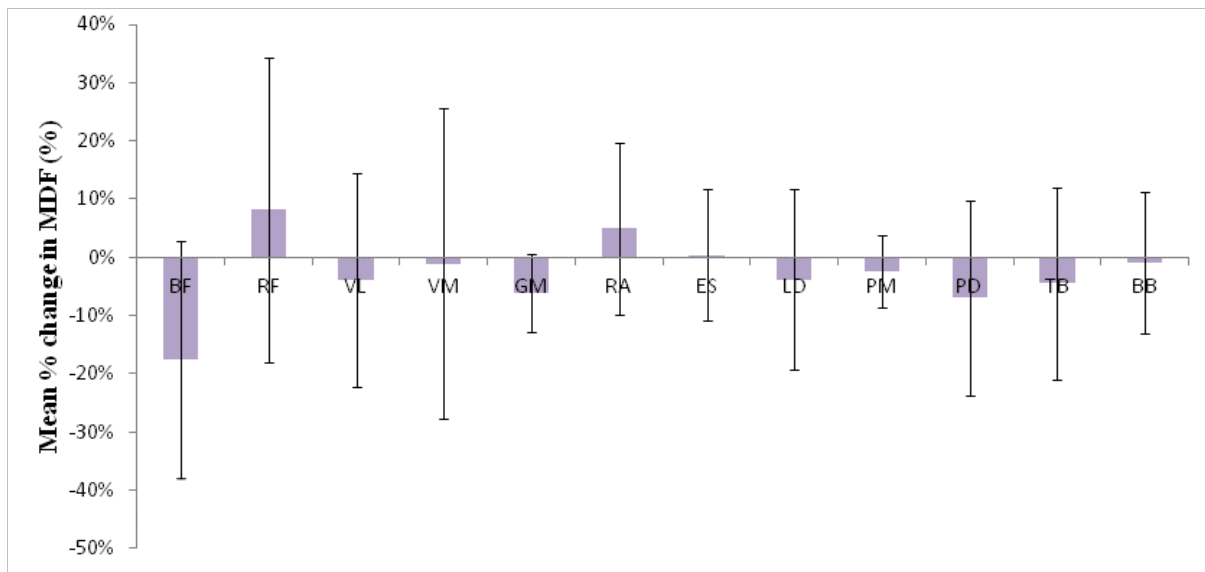


**Figure 5.11** A comparison, between each muscle, of the mean percentage change in mean power frequency (MPF) over the duration of the 100 m breaststroke swim: *BF*, biceps femoris; *RF*, rectus femoris; *VL*, vastus lateralis; *VM*, vastus medialis; *GM*, gluteus maximus; *RA*, rectus abdominals; *ES*, erectus spinae; *LD*, latissimus dorsi; *PM*, pectoralis major; *PD*, posterior deltoid; *TB*, triceps brachii; *BB*, biceps brachii. Data represent mean  $\pm$  SD.

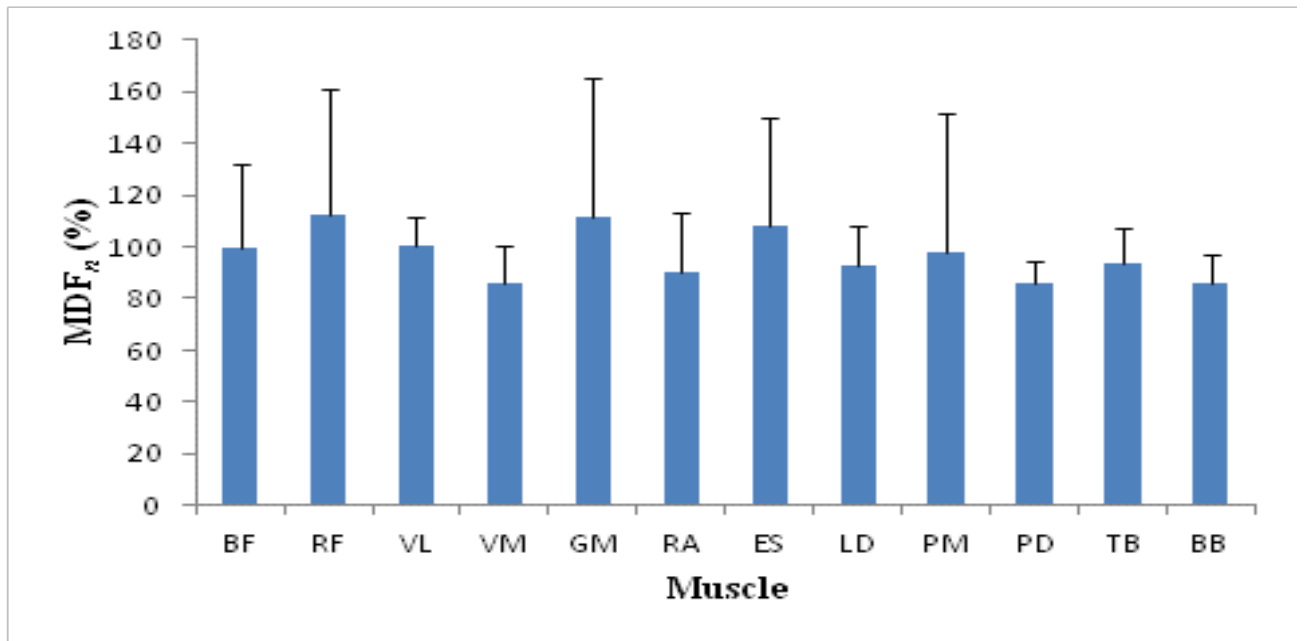
Ten of the twelve muscles showed a decrease in mean power frequency (MPF) over the duration of the swim with two of the muscles showing an overall increase in MPF (Figure 5.11). Overall ten of the twelve muscles showed a decrease in median power frequency (MDF) over the duration of the swim and two of the muscle showed an overall increase in MDF (Figure 5.12). Over the duration of the whole swim for the muscles of the lower limb there was a mean increase in MPF of 10% in rectus femoris compared to a mean decrease in vastus lateralis (8%) and vastus medialis (5%) (Figure 5.11).

Over the duration of the whole swim for the muscles of the trunk the muscle gluteus maximus showed a 2% decrease in mean MDF with a similar 1% decrease in erectus spinae, however rectus abdominal showed an overall increase in MDF of 2% (Figure 5.12). Over the duration of the swim rectus abdominal showed an 8 % increase in MPF compared to an overall decrease of 2% in gluteus maximus and an overall decrease of 3% in erectus spinae (Figure 5.11).

The overall changes in MDF for the duration of the whole swim for the muscles of the upper limb showed that the latissimus dorsi and pectoralis major decreased by 5% and 3% respectfully (Figure 5.12). The other upper body muscles, posterior deltoid (7%), triceps brachii (4%) and biceps brachii (1%), showed overall decreases in MDF over the duration of the swim (Figure 5.12). Over the duration of the whole swim there was a mean decrease in MPF in the muscles (Figure 5.11). The muscle posterior deltoid (8%) showed the largest overall decrease in MPF

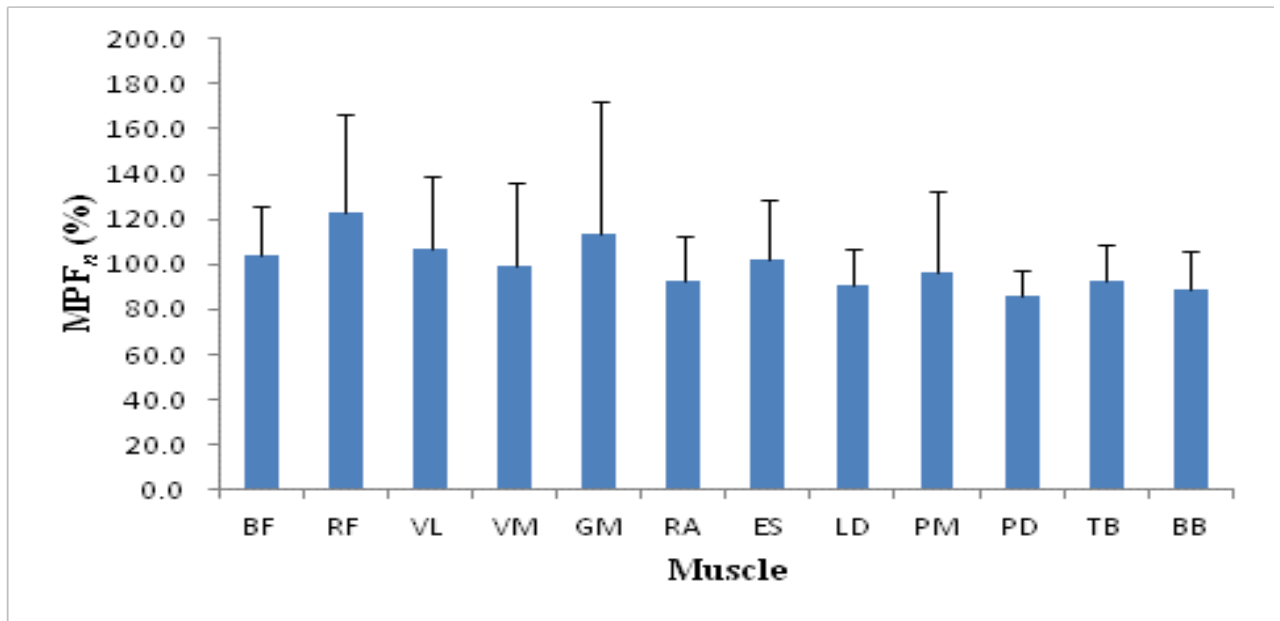


**Figure 5.12** A comparison, between each muscle, of mean percentage change in median frequency (MDF) over the duration of the 100 m breaststroke swim: *BF*, biceps femoris; *RF*, rectus femoris; *VL*, vastus lateralis; *VM*, vastus medialis; *GM*, gluteus maximus; *RA*, rectus abdominals; *ES*, erectus spinae; *LD*, latissimus dorsi; *PM*, pectoralis major; *PD*, posterior deltoid; *TB*, triceps brachii; *BB*, biceps brachii. Data represent mean  $\pm$  SD.



**Figure 5.13** Normalised  $MDF_n$  (%) at the end of swimming for each muscle *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis*; *GM*, *gluteus maximus*; *RA*, *rectus abdominals*; *ES*, *erectus spinae*; *LD*, *latissimus dorsi*; *PM*, *pectoralis major*; *PD*, *posterior deltoid*; *TB*, *triceps brachii*; *BB*, *biceps brachii*. Data represent mean  $\pm$  SD.

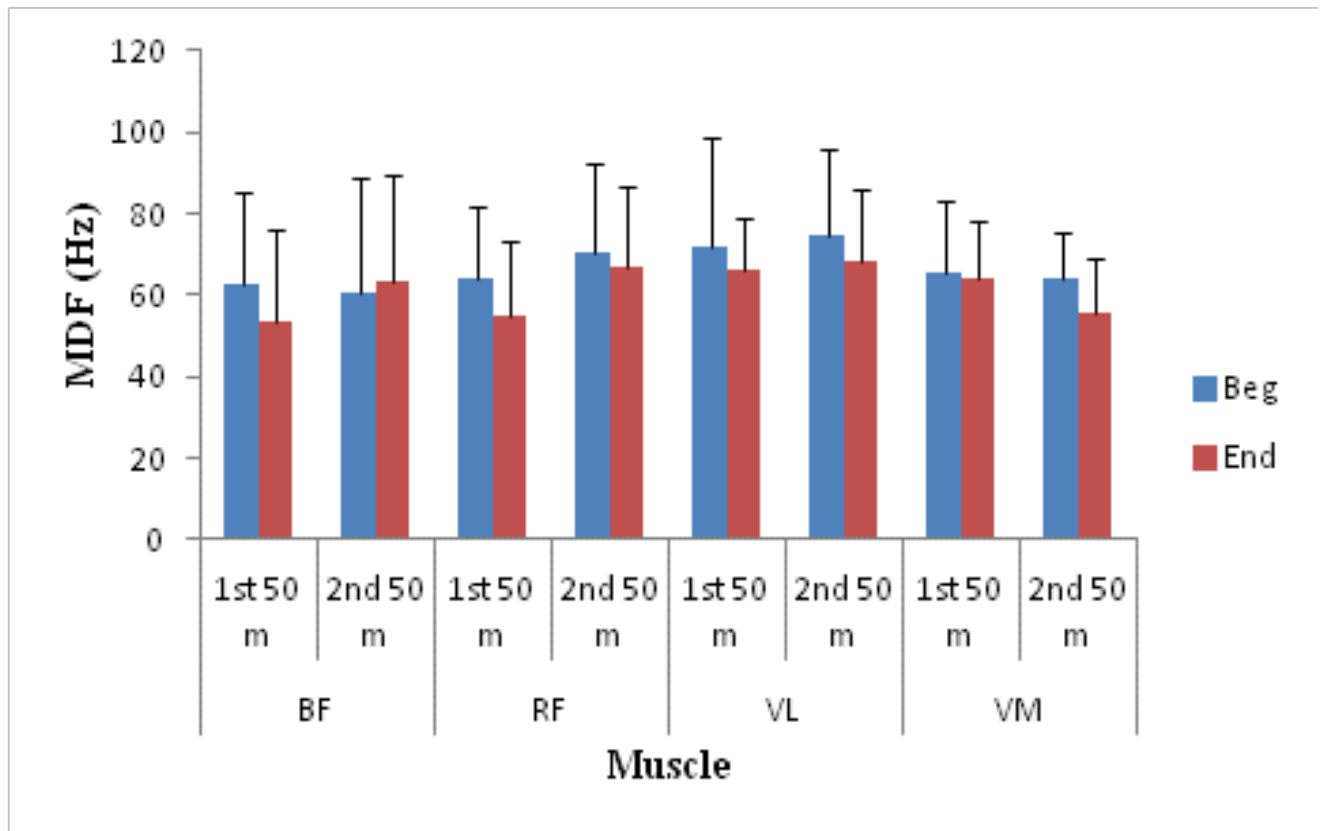
When the data were normalised and the relative decrease from the beginning of the 1<sup>st</sup> 50 m to the end of the 2<sup>nd</sup> 50 m of the swim were analysed  $MPF_n$  showed no statistically significant difference between any of the twelve muscles  $F(11,70) 0.86, p = .579, \eta^2 0.12$  (Figure 5.13). There was also no statistically significant difference in normalised  $MDF_n$  at the end of the swim between muscles  $F(11,70) 0.62, p = .804, \eta^2 0.09$  (Figure 5.14).



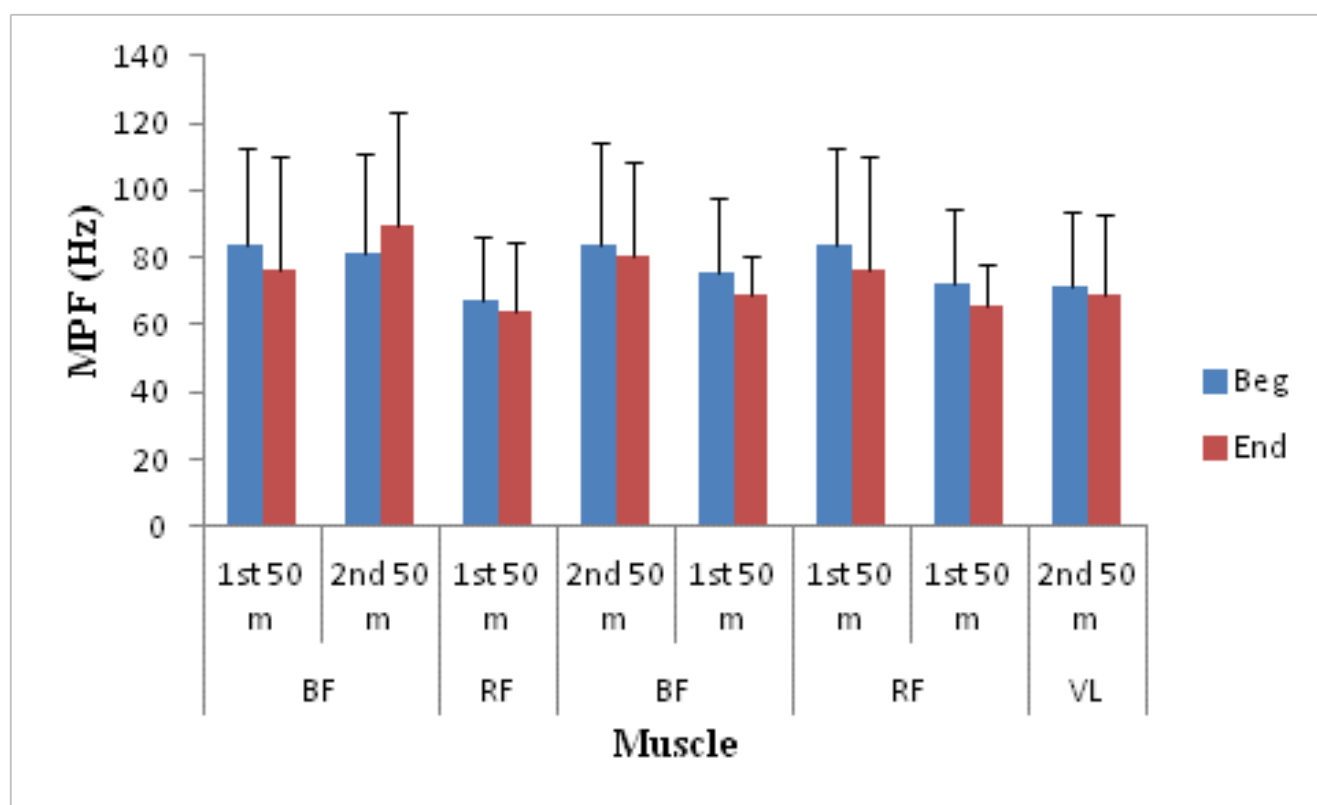
**Figure 5.14** Normalised MPF<sub>n</sub> (%) at the end of swimming for each muscle *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis*; *GM*, *gluteus maximus*; *RA*, *rectus abdominals*; *ES*, *erectus spinae*; *LD*, *latissimus dorsi*; *PM*, *pectoralis major*; *PD*, *posterior deltoid*; *TB*, *triceps brachii*; *BB*, *biceps brachii*. Data represent mean  $\pm$  SD.



#### 5.4.4 EMG Changes in the Muscles of the Leg

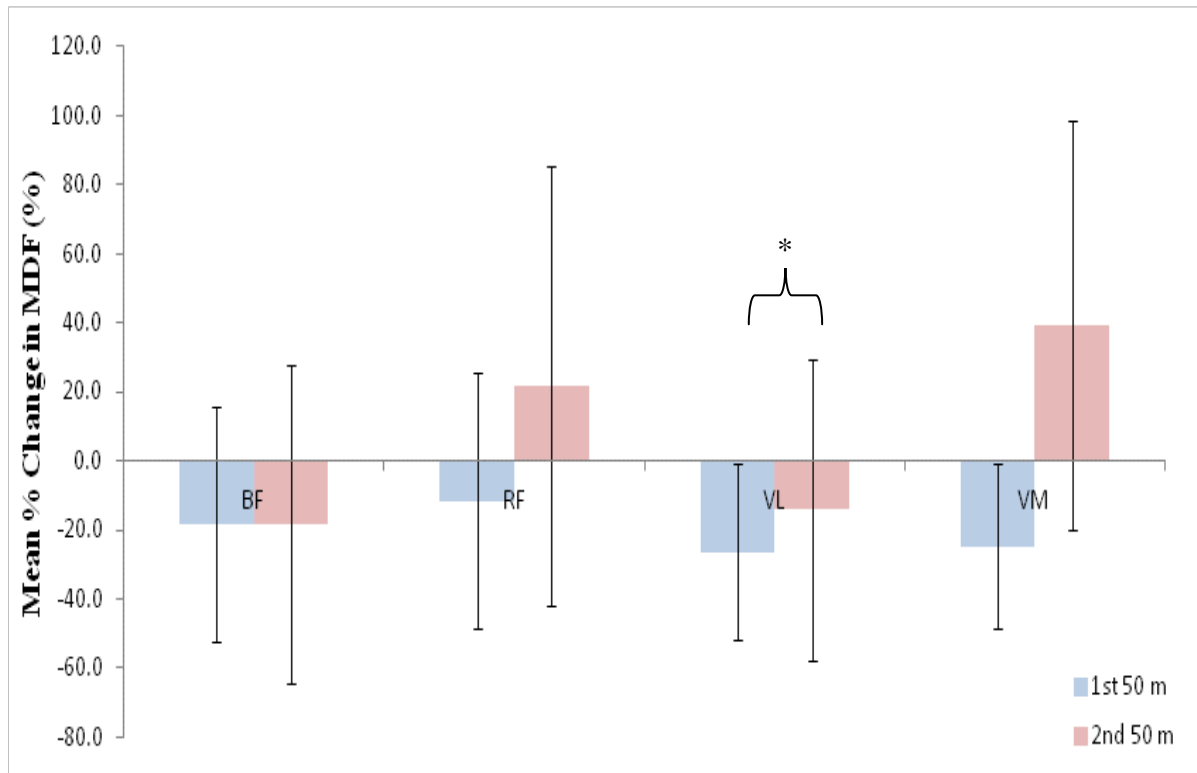


**Figure 5.15** The mean MDF value with SD for  $MDF_{Beg}$  and  $MDF_{End}$  (Blue and Red filled bars respectfully) for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m shown for each of the muscles: *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis* muscles of the lower body. Data represent mean  $\pm$  SD.

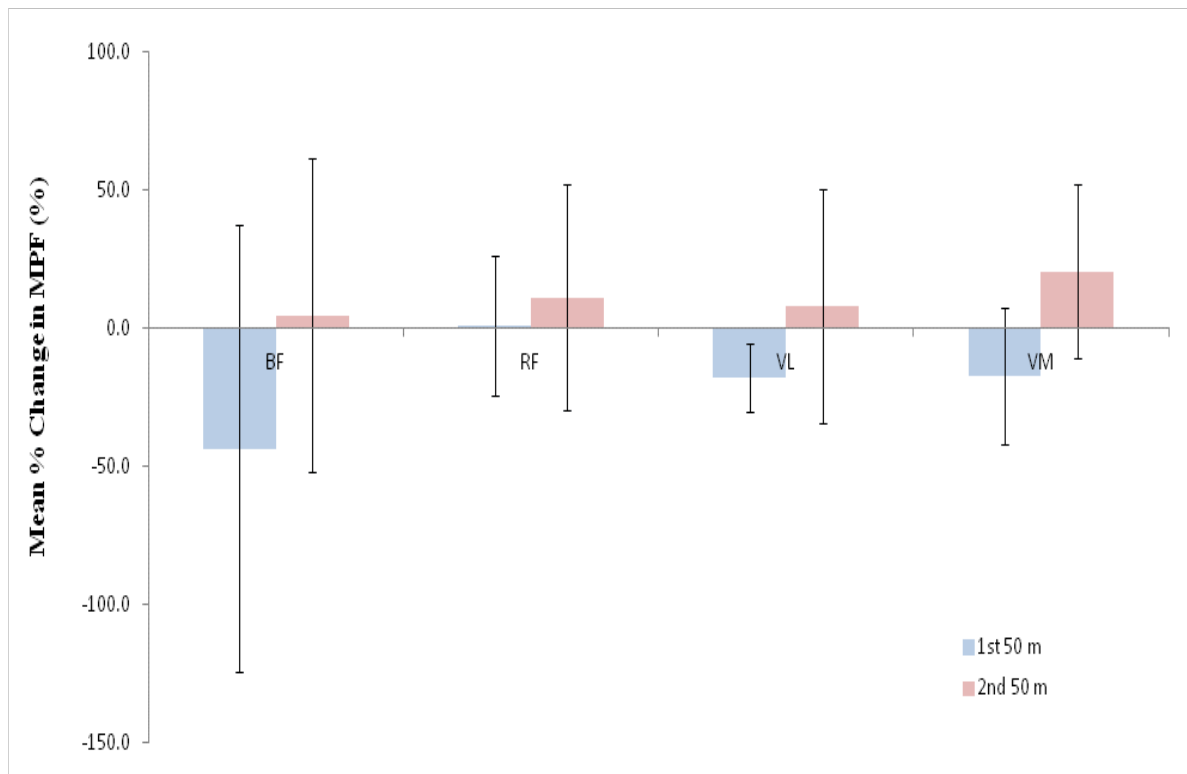


**Figure 5.16** The mean MPF value with SD for  $MPF_{Beg}$  and  $MPF_{End}$  (Blue and Red filled bars respectfully) for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m shown for each of the muscles: *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis* muscles of the lower body. Data represent mean  $\pm$  SD.

Analysis of variance for each muscle of the lower limb showed no statistically significant difference between the mean MDF (Figure 5.15) at the beginning ( $MDF_{Beg}$ ) or end ( $MDF_{End}$ ) of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m ( $p > 0.05$ ). There was also no significant difference reported in MPF at the beginning ( $MPF_{Beg}$ ) or end ( $MPF_{End}$ ) of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m ( $p > 0.05$ ) (Figure 5.16).

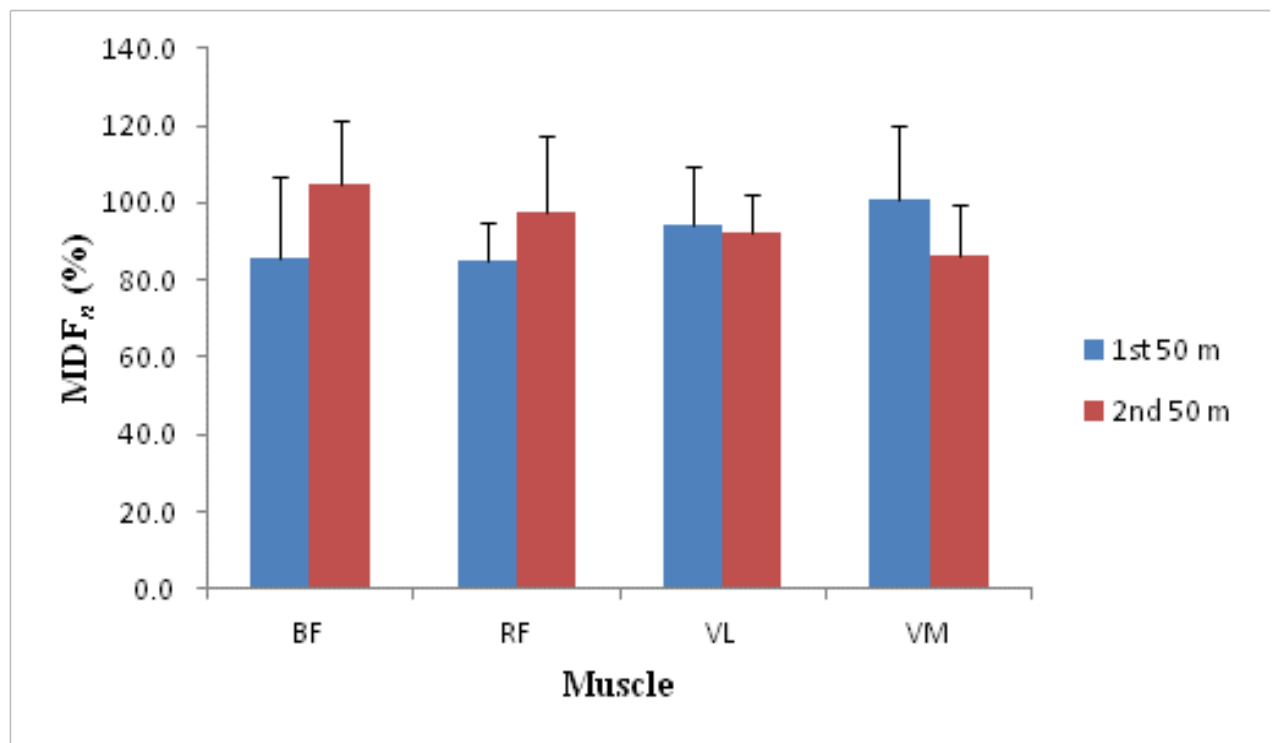


**Figure 5.17** A comparison of the mean percentage change in median frequency (MDF) during the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m breaststroke swim: *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis* muscles of the lower body. Data represent mean  $\pm$  SD. Note the significant difference between the 1<sup>st</sup> 50 m and the 2<sup>nd</sup> 50 m for VL (\* $p < 0.05$ ).

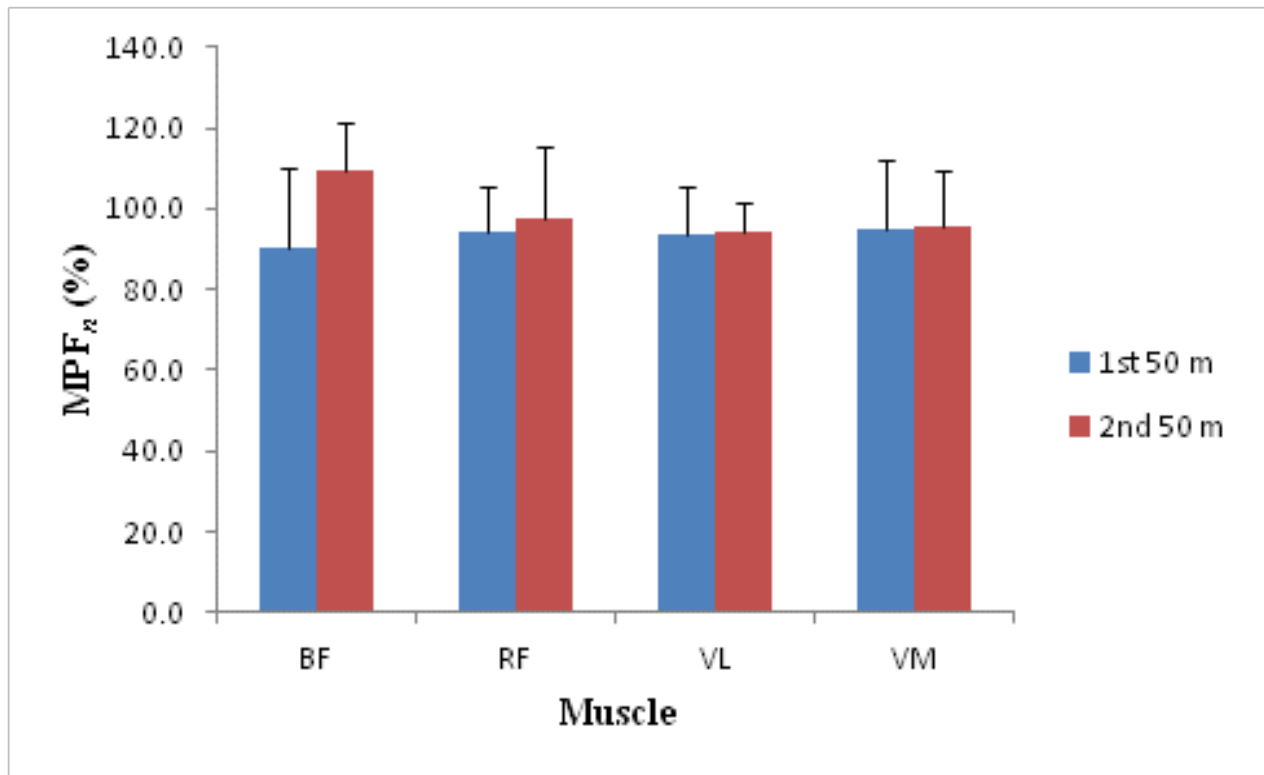


**Figure 5.18** A comparison of the mean percentage change in mean power frequency (MPF) during the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of each muscle: *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis*; of the lower body. Data represent mean  $\pm$  SD.

The rate of change in MDF of the muscles of the lower limb showed that the only muscle that significantly changes from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m was the muscle vastus lateralis  $t(6) -2.85, p = .029, r = 0.75$ , (Figure 5.17) with a % decrease in MDF. The remaining muscles of the lower limb showed no significant change in MDF from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m ( $p > 0.05$ ). The rate of change for MPF showed no statistically significant changes from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m ( $p > 0.05$ , Figure 5.18)



**Figure 5.19** Normalised  $MDF_n$  (%) at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m (blue and red filled bars respectfully) shown for each of the muscles: *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis* muscles of the lower body. Data represent mean  $\pm$  SD.

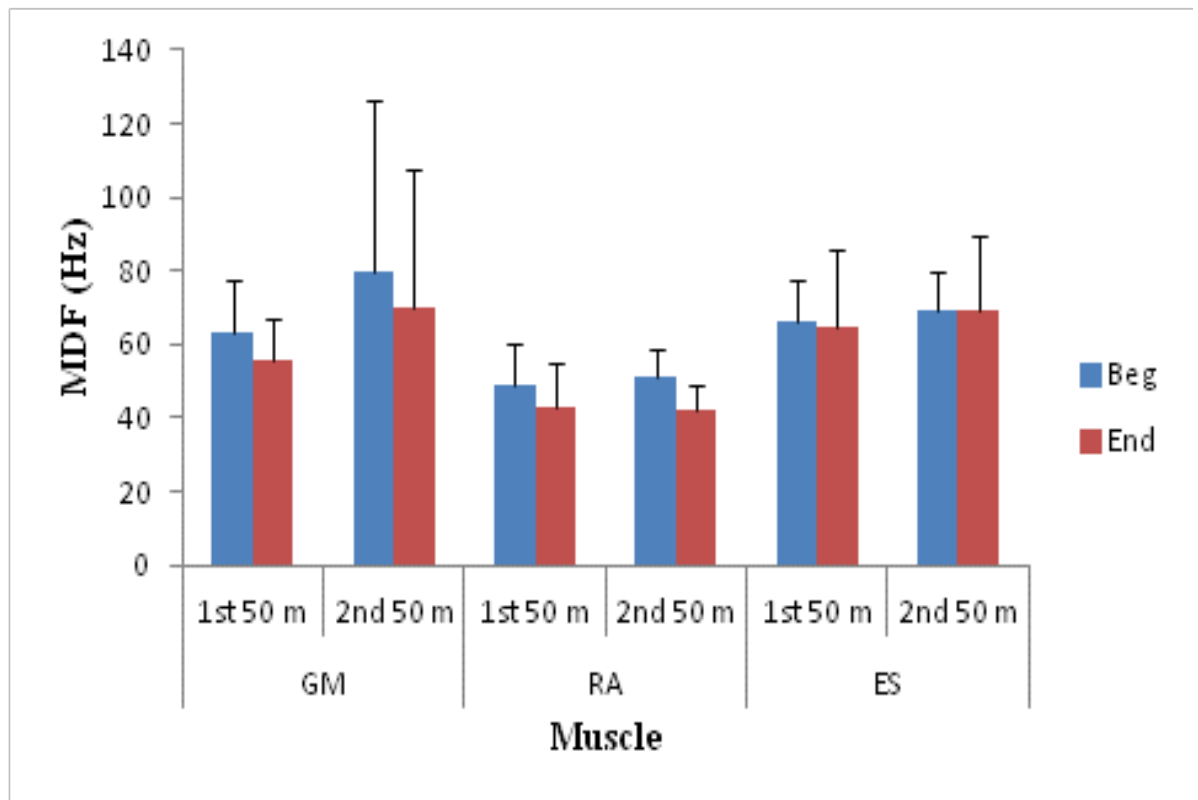


**Figure 5.20** Normalised  $MPF_n$  (%) at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m (blue and red filled bars respectfully) shown for each of the muscles: *BF*, *biceps femoris*; *RF*, *rectus femoris*; *VL*, *vastus lateralis*; *VM*, *vastus medialis* muscles of the lower body. Data represent mean  $\pm$  SD.

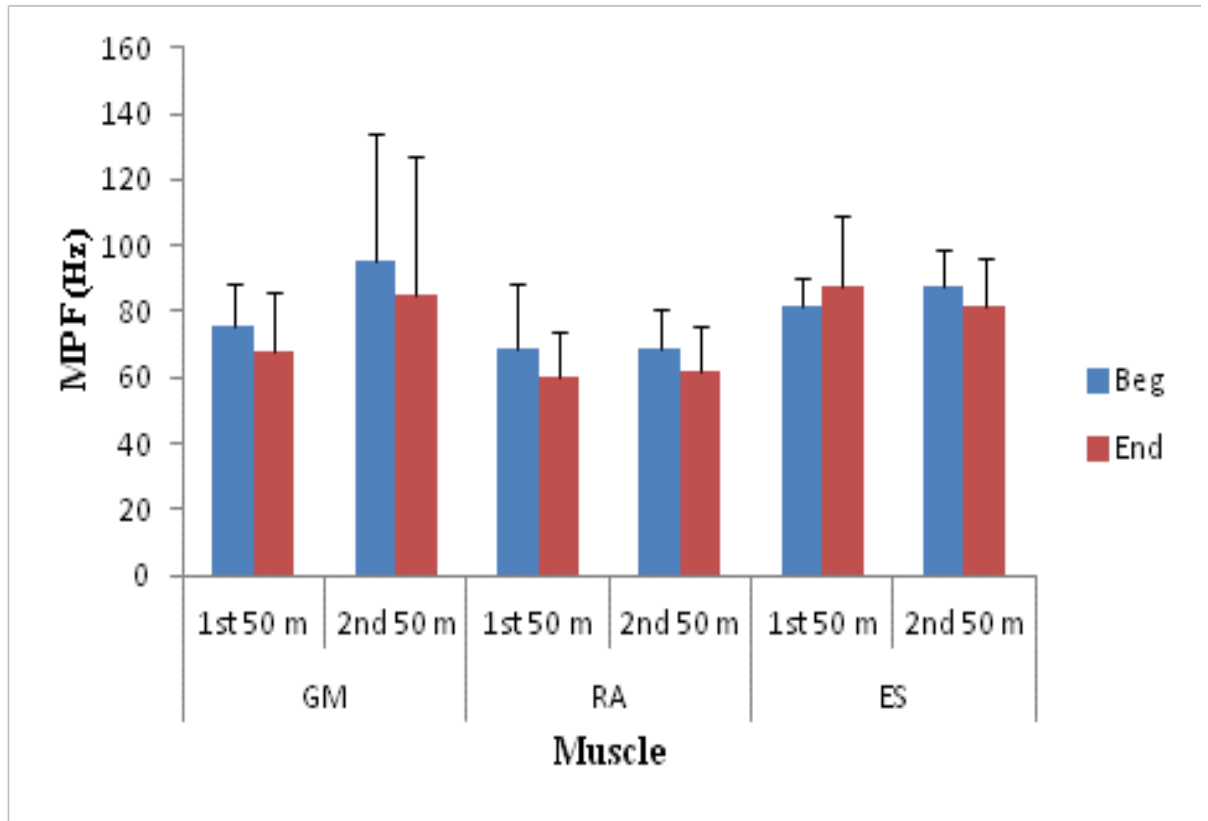
Analysis of variance of the normalised  $MDF_n$  at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for the muscles of the lower limb showed no significant main effect for muscle  $F(3,36)$  0.15,  $p = .930$ ,  $\eta^2$  0.01 (Figure 5.9). There was no main effect between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m  $F(1,12)$  0.87,  $p = .37$ ,  $\eta^2$  0.07 (Figure 5.9). There was no significant interaction between muscle and lap although it did approach statistical significance  $F(3,36)$  2.85,  $p = .051$ ,  $\eta^2$  0.19. Analysis of variance of the normalised  $MPF_n$  at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for the muscles of the lower limb showed no significant main effect for muscle  $F(3,36)$  0.45,  $p = .72$ ,  $\eta^2$  0.04 (Figure 5.10). There was no significant difference between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for  $MPF_n$ .

$F(1,12) 1.78, p = .207, \eta p^2 0.13$  (Figure 5.10). There was no significant interaction between muscle and the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for  $MPF_n F(3,36) 1.48, p = .237, \eta p^2 0.11$ .

#### 5.4.5 EMG Changes in Muscles of the Hip and Trunk



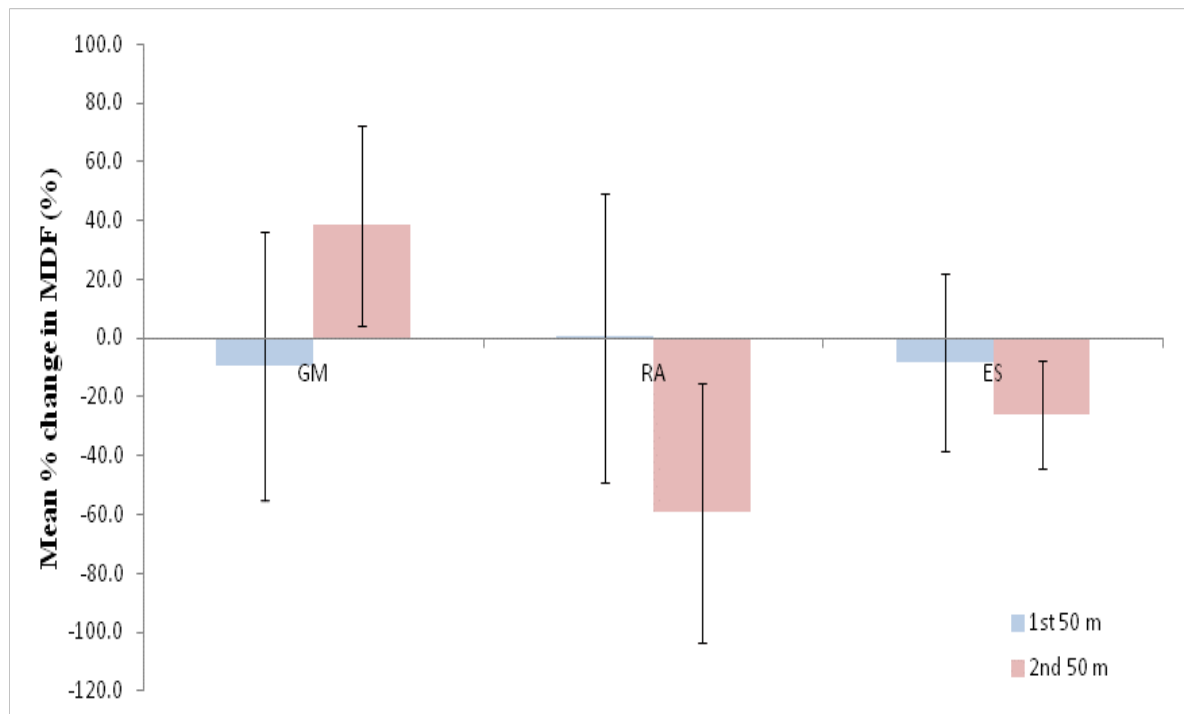
**Figure 5.21** The mean MDF value with SD for  $MDF_{Beg}$  and  $MDF_{End}$  (Blue and Red filled bars respectfully) for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m shown for each of the muscles: *GM*, *gluteus maximus*; *RA*, *rectus abdominals*; *ES*, *erectus spinae*; muscles of the hips and trunk. Data represent mean  $\pm$  SD.



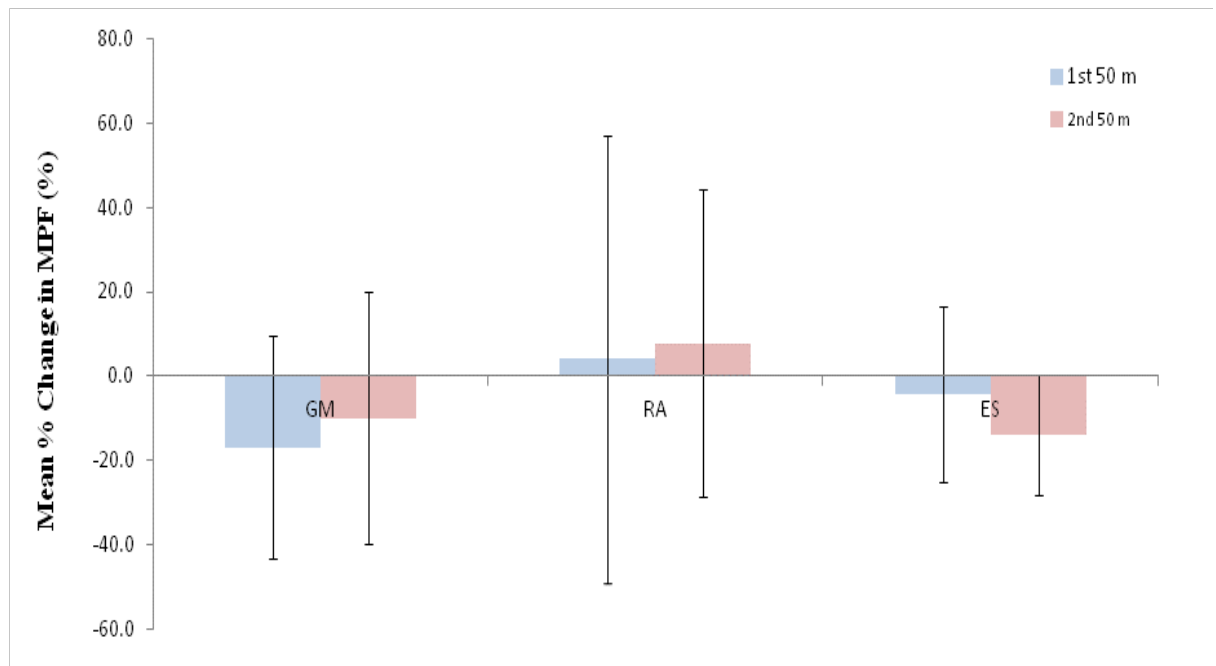
**Figure 5.22** The mean MPF value with SD for MPF<sub>Beg</sub> and MPF<sub>End</sub> (Blue and Red filled bars respectfully) for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m shown for each of the muscles: *GM*, *gluteus maximus*; *RA*, *rectus abdominals*; *ES*, *erectus spinae*; muscles of the hips and trunk. Data represent mean  $\pm$  SD.

Analysis of variance for each muscle of the trunk showed no statistically significant difference between the mean MDF (Figure 5.21) at the beginning (MDF<sub>Beg</sub>) or end (MDF<sub>End</sub>) of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m ( $p > 0.05$ ). There was also no significant difference reported in MPF ( $p > 0.05$ , Figure 5.22). Overall there was no statistically significant difference in rate of change in MDF (Figure 5.23) or in MPF (Figure 5.24) between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for any of the muscles of the hip and trunk ( $p > 0.05$ )

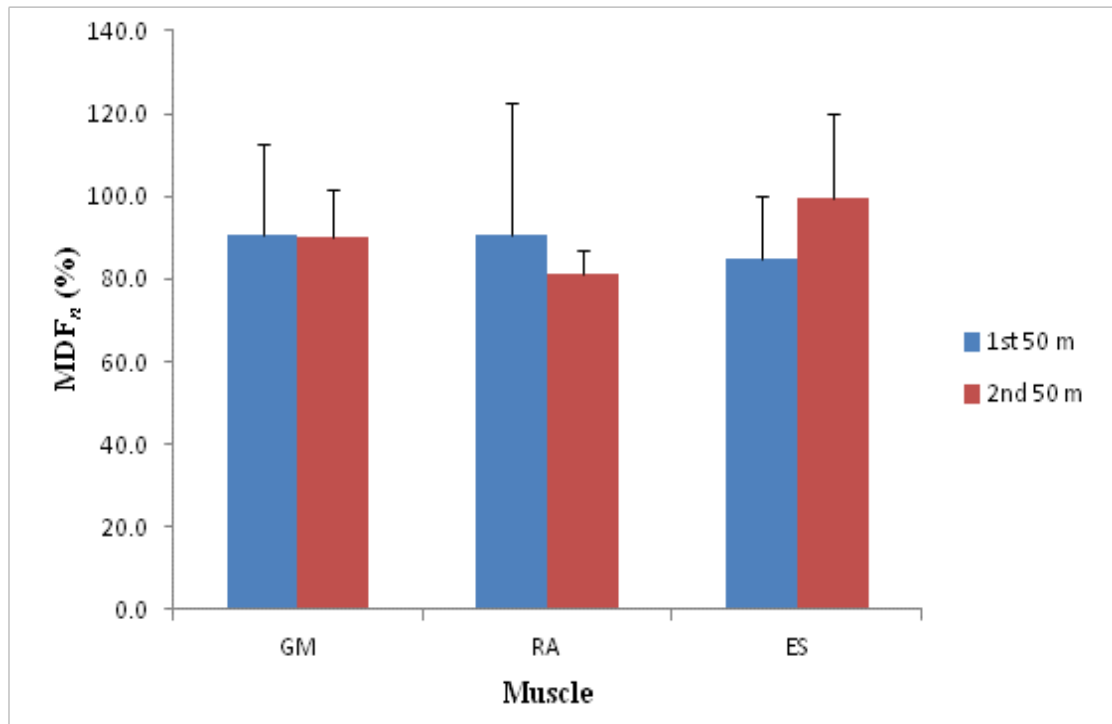




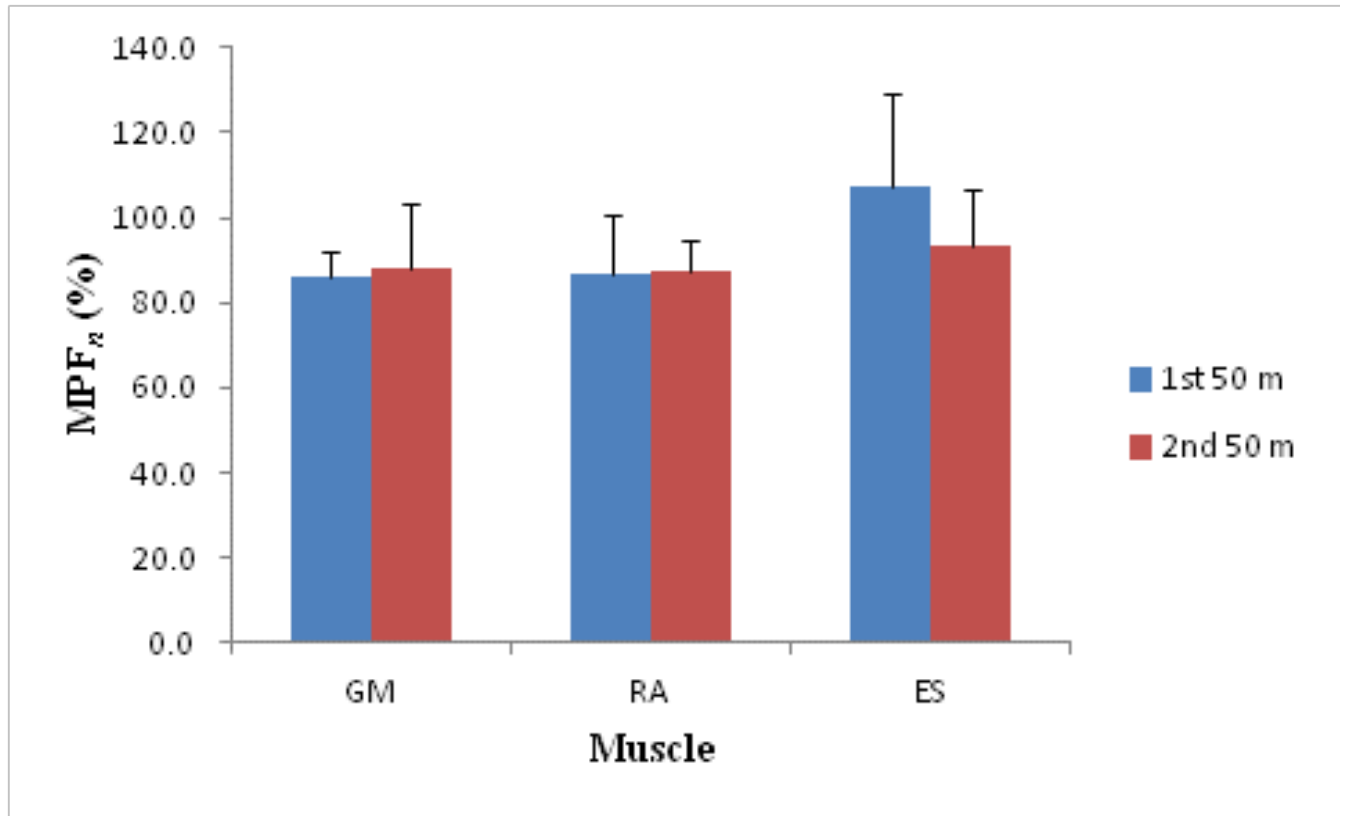
**Figure 5.23** A comparison of the mean percentage change in median frequency (MDF) during the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m breaststroke swim: *GM*, *gluteus maximus*; *RA*, *rectus abdominals*; *ES*, *erectus spinae*; muscles of the hips and trunk. Data represent mean  $\pm$  SD.



**Figure 5.24** A comparison of the mean percentage change in mean power frequency (MPF) during the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m breaststroke swim: *GM*, *gluteus maximus*; *RA*, *rectus abdominals*; *ES*, *erectus spinae* muscles of the hips and trunk. Data represent mean  $\pm$  SD.



**Figure 5.25** Normalised  $MDF_n$  (%) at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m (blue and red filled bars respectfully) shown for each of the muscles: *GM*, *gluteus maximus*; *RA*, *rectus abdominals*; *ES*, *erectus spinae*; muscles of the hips and trunk. Data represent mean  $\pm$  SD.

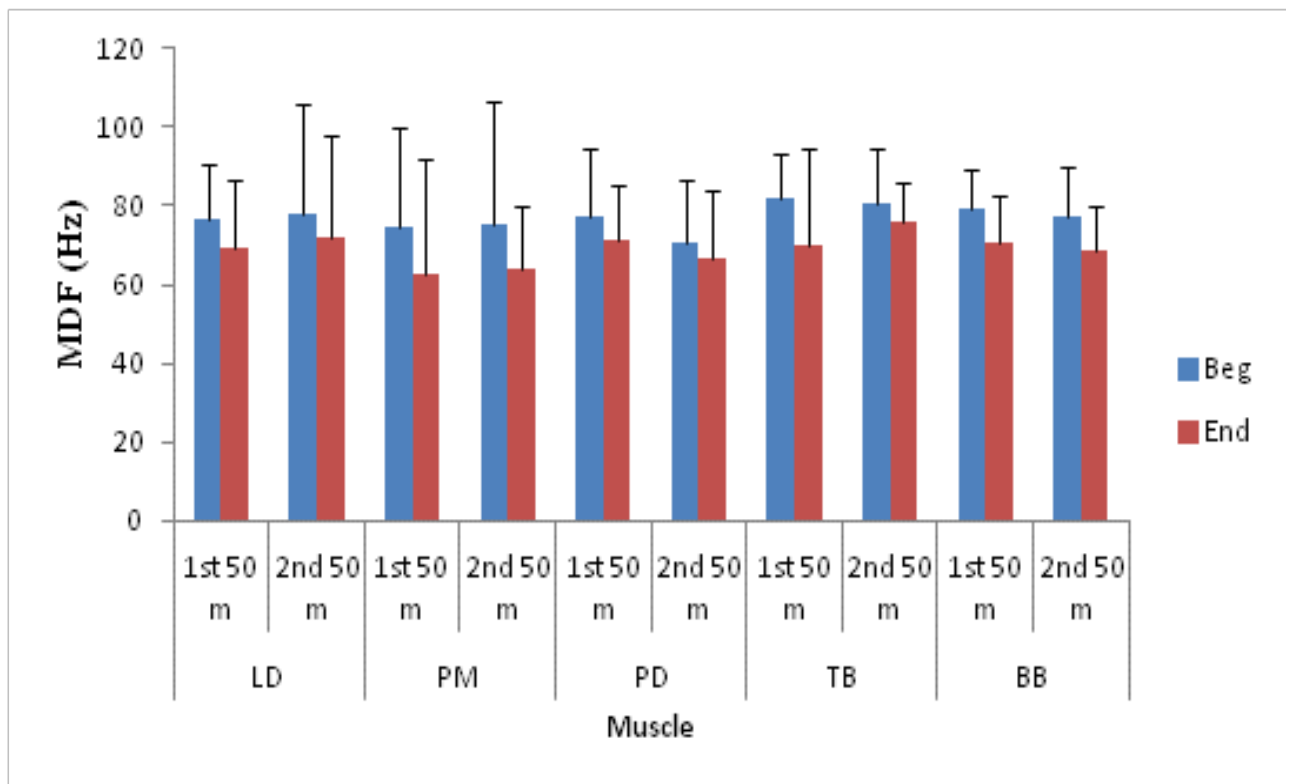


**Figure 5.26** Normalised MPF<sub>n</sub> (%) at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m (blue and red filled bars respectfully) shown for each of the muscles: *GM*, *gluteus maximus*; *RA*, *rectus abdominals*; *ES*, *erectus spinae*; muscles of the hips and trunk. Data represent mean  $\pm$  SD.

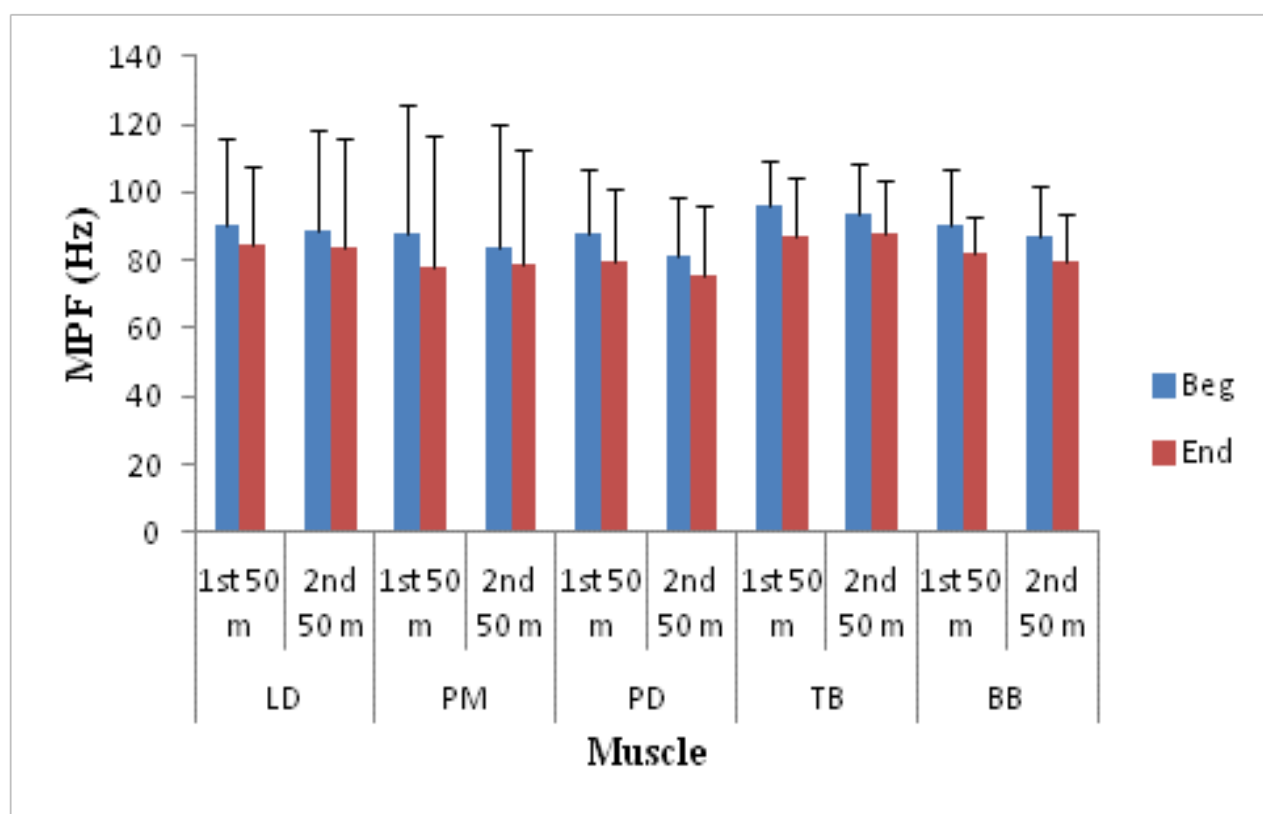
Analysis of variance of the normalised MDF<sub>n</sub> at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for the muscles of the trunk showed no significant main effect for muscle  $F(2,16)$  1.04,  $p = .378$ ,  $\eta^2$  0.12 (Figure 5.25). There was no main effect between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m  $F(1,8)$  2.74,  $p = .137$ ,  $\eta^2$  0.26 (Figure 5.26). There was no significant interaction between muscle and lap  $F(2,16)$  0.34,  $p = .719$ ,  $\eta^2$  0.04. Analysis of variance of the normalised MPF<sub>n</sub> at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for the muscles of the trunk showed no significant main effect for muscle although it approach statistical significance  $F(2,16)$  3.60,  $p = .051$ ,  $\eta^2$  0.31 (Figure 5.14). There was no statistically significant change between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for MPF<sub>n</sub>  $F(1,8)$

0.02,  $p = .900$ ,  $\eta p^2$  0.002 (Figure 5.14). There was no significant interaction between muscle and the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for  $MPF_n F(2,16)$  0.15,  $p = .864$ ,  $\eta p^2$  0.02.

#### 5.4.6 EMG changes in Muscle of the Upper Trunk

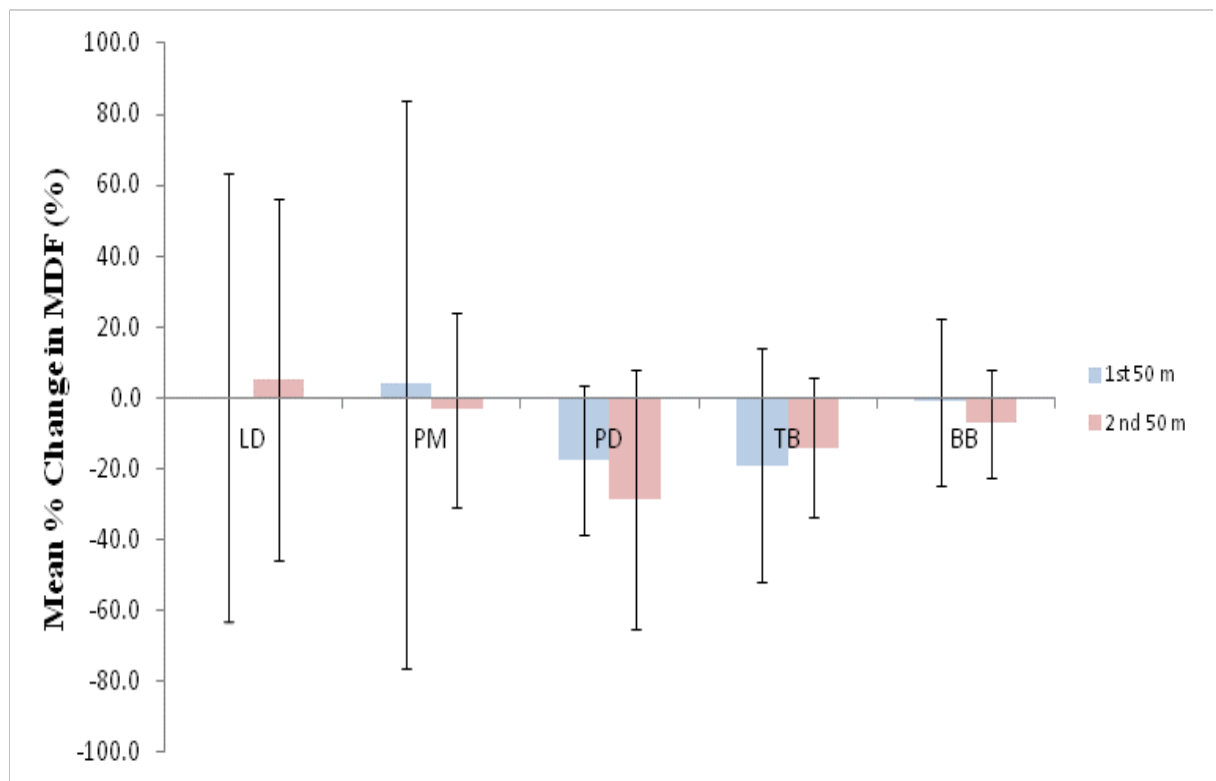


**Figure 5.27** The mean MDF value with SD for  $MDF_{Beg}$  and  $MDF_{End}$  (Blue and Red filled bars respectfully) for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m shown for each of the muscles: *LD*, *latissimus dorsi*; *PM*, *pectoralis major*; *PD*, *posterior deltoid*; *TB*, *triceps brachii*; *BB*, *biceps brachii* muscles of the upper trunk. Data represent mean  $\pm$  SD.

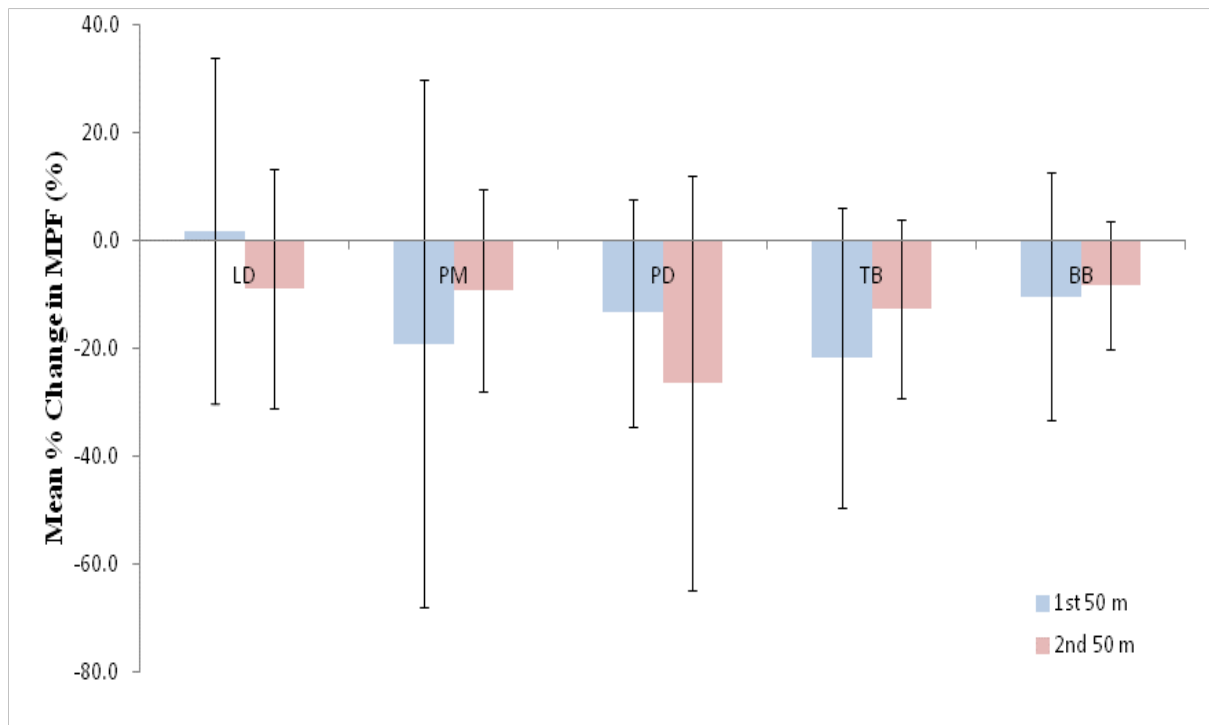


**Figure 5.28** The mean MPF value with SD for MPF<sub>Beg</sub> and MPF<sub>End</sub> (Blue and Red filled bars respectfully) for the 1<sup>st</sup> and 2<sup>nd</sup> 50 m shown for each of the muscles: *LD*, *latissimus dorsi*; *PM*, *pectoralis major*; *PD*, *posterior deltoid*; *TB*, *triceps brachii*; *BB*, *biceps brachii* muscles of the upper trunk. Data represent mean  $\pm$  SD.

Analysis of variance for each muscle of the upper limb showed no statistically significant difference between the mean MDF (Figure 5.27) at the beginning (MDF<sub>Beg</sub>) or end (MDF<sub>End</sub>) of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m  $p > 0.05$  or for mean MPF at the beginning or end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m ( $p > 0.05$ , Figure 5.28). Overall there was no statistically significant difference in the rate of change for MDF (Figure 5.29) or MPF (Figure 5.30) between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for any of the muscles of the upper body ( $p > 0.05$ ).

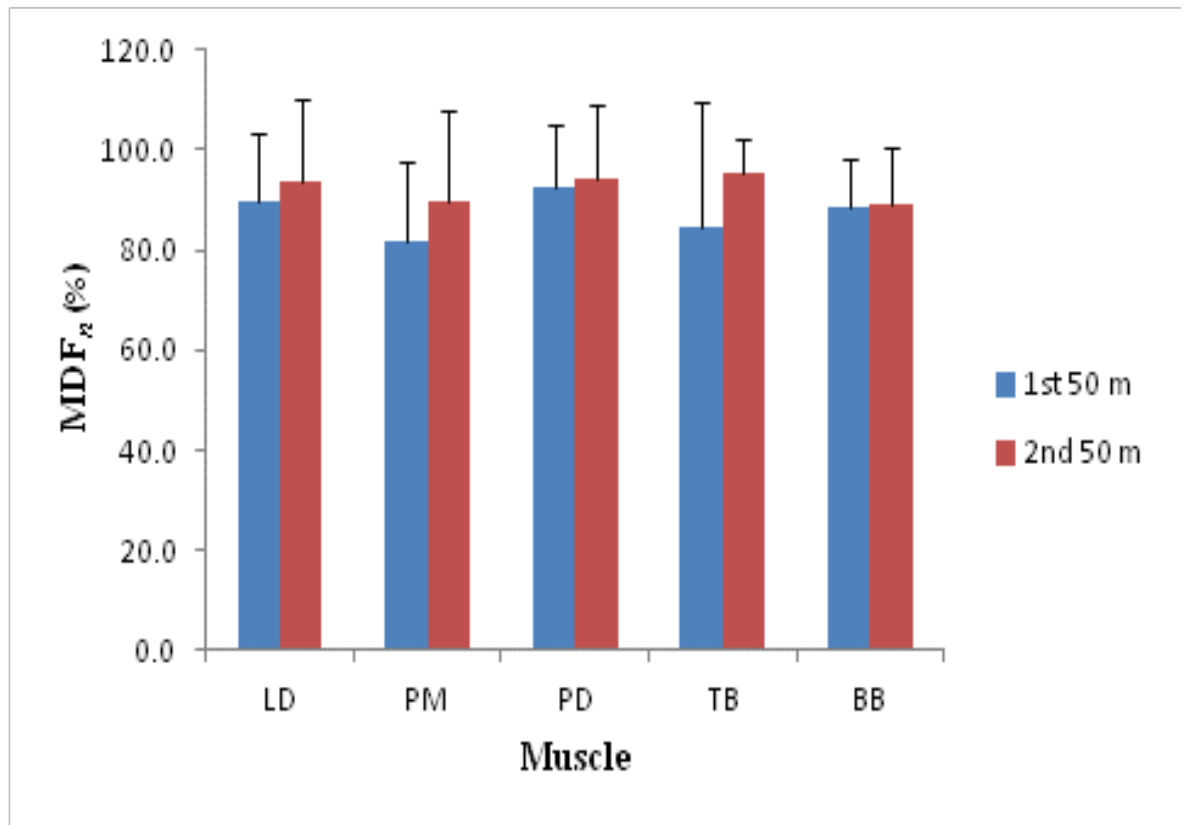


**Figure 5.29** A comparison of the mean percentage change in median frequency (MDF) during the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m breaststroke swim: *LD*, *latissimus dorsi*; *PM*, *pectoralis major*; *PD*, *posterior deltoid*; *TB*, *triceps brachii*; *BB*, *biceps brachii*; muscles of the upper trunk. Data represent mean  $\pm$  SD.

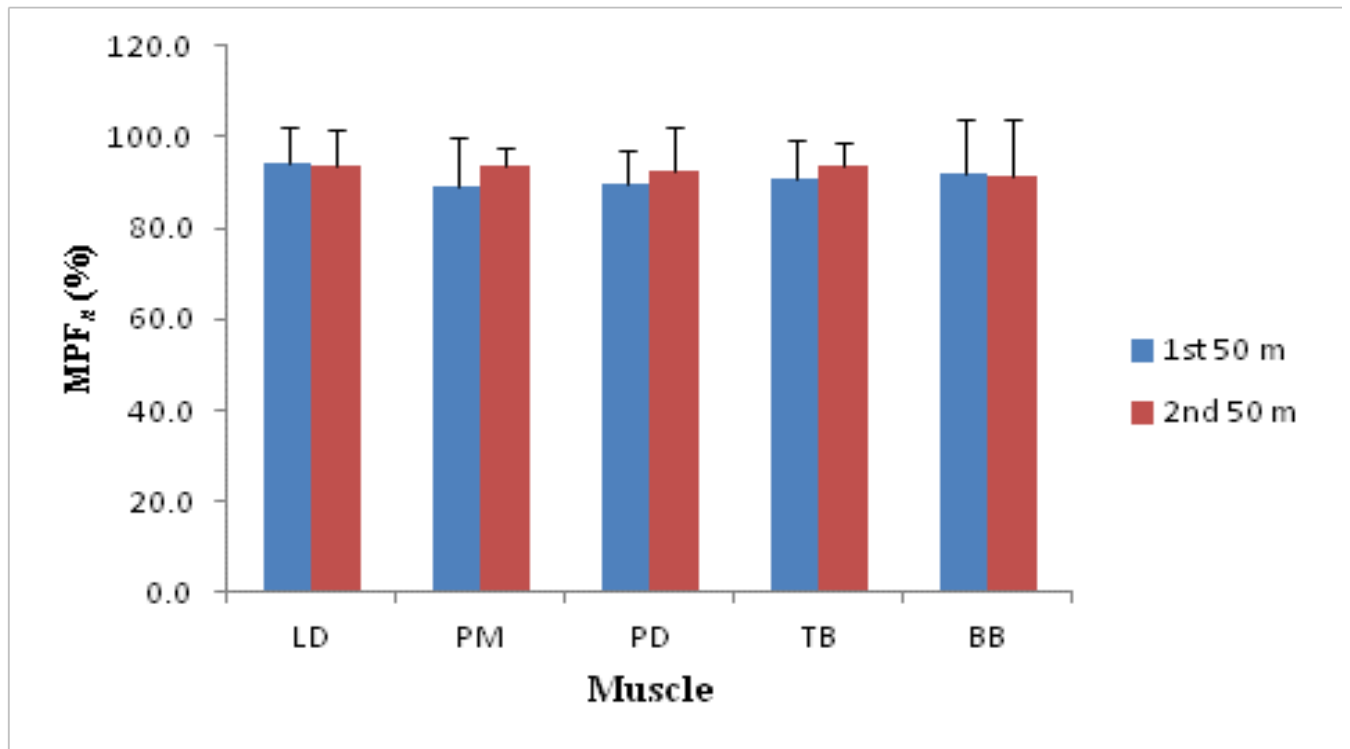


**Figure 5.30** A comparison of the mean percentage change in mean power frequency (MPF) during the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m breaststroke swim: *LD*, *latissimus dorsi*; *PM*, *pectoralis major*; *PD*, *posterior deltoid*; *TB*, *triceps brachii*; *BB*, *biceps brachii* muscles of the upper trunk. Data represent mean  $\pm$  SD.





**Figure 5.31** Normalised  $MDF_n$  (%) at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m (blue and red filled bars respectfully) shown for each of the muscles: *LD*, *latissimus dorsi*; *PM*, *pectoralis major*; *PD*, *posterior deltoid*; *TB*, *triceps brachii*; *BB*, *biceps brachii* muscles of the upper trunk. Data represent mean  $\pm$  SD.



**Figure 5.32** Normalised MPF<sub>n</sub> (%) at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m (blue and red filled bars respectfully) shown for each of the upper trunk muscles: *LD*, *latissimus dorsi*; *PM*, *pectoralis major*; *PD*, *posterior deltoid*; *TB*, *triceps brachii*; *BB*, *biceps brachii* muscles of the upper trunk. Data represent mean  $\pm$  SD.

Analysis of variance of the normalised MDF<sub>n</sub> at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for the muscles of the upper trunk showed no significant main effect for muscle  $F(4,48) 0.707, p = .591, \eta^2 0.06$  (Figure 5.31). There was no main effect between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m  $F(1,12) 0.97, p = .343, \eta^2 0.08$  (Figure 5.31). There was no significant interaction between muscle and lap  $F(4,48) 0.36, p = .833, \eta^2 0.03$ . Analysis of variance of the normalised MPF<sub>n</sub> at the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for the muscles of the upper trunk showed no significant main effect for muscle  $F(4,48) 0.20, p = .939, \eta^2 0.02$  (Figure 5.32). There was no statistically significant change between the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for MPF<sub>n</sub>  $F(1,12) 0.50, p = .494, \eta^2 0.04$

(Figure 5.32). There was no significant interaction between muscles of the upper trunk and the 1<sup>st</sup> and 2<sup>nd</sup> 50 m for MPF<sub>n</sub>  $F(4,48)$  0.24,  $p = .915$ ,  $\eta p^2$  0.02.

## 5.5 Discussion

### 5.5.1 Kinematics

Swimmers have been shown to adopt specific combinations of stroke length (SL) and stroke rate (SR) that change over race distance, presumably as a consequence of fatigue (Thompson, Haljand and MacLaren, 2000). This was the same for the current study where the variability in the combinations of the SL and SR was showed (Table 5.1) indicating high levels of inter subject variability. The participant's combination of their SL and SR does not remain constant from the 1<sup>st</sup> to 2<sup>nd</sup> 50 m as shown by the decrease in SL from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. In the current study there was a 7.9% ( $p = .032$ ) decrease in SL, from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m, which is similar to the 8 % decrease reported by Thompson, et al., (2000) in breaststroke swimming. This finding thus supports hypothesis 1. A decrease in SL has been reported to be the first sign of fatigue in swimmers (Dekerle et al., 2005; Keskinen and Komi, 1993; Weiss et al., 1988). This suggests that each individual responds differently to try and maintain clean swim speed as they progress through a swim and may differ in their rate of fatigue.

As a consequence of a decrease in SL, swimmers have to try and increase SR in an attempt to maintain clean swim speed (Thompson, Haljand and MacLaren, 2000). This is not confirmed by the findings of the current study as there was no significant change in SR from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m of the swim (Table 4.1). As there was no increase in SR there was

nothing to offset the decrease in SL, resulting in an overall decrease of 6.4% in the mean clean swim speed from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m (Table 4.1), which is in line with the previously reported decrease of 6 -7 % in breaststroke swimming (Thompson, Haljand and MacLaren, 2000). Our findings contrast to those of Seifert, et al., (2009) who reported that front crawl swimmers change speed through alterations in SR and not via alterations in their co-ordination. In the current study the findings suggest that there were no significant ( $p < 0.05$ ) alterations in the participants' co-ordination patterns from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m (Table 4.2). Overall there was no significant change in co-ordination phase 1 (Table 4.2) from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. This may be due to the high levels of inter-subject variability in the co-ordination of the propulsive phases of the arms and legs thus rejecting hypothesis 4. The co-ordination phase 1 time was characterised as the time between the end of the leg propulsion phase and the start of the arm propulsion phase (Figure 2.1, Section 2.3.4, Chapter 2). Four of the seven participants started with the glide overlap on the first lap and the remainder of the participants utilised the glide co-ordination technique. These findings are in line with those in chapter 2 (Oxford et al., 2010) that the majority of participants showed that their co-ordination pattern changed in the same direction to that of overlap or greater overlap with in the stroke cycle.

Fatigue has been defined as a failure to maintain power output (Edwards, 1983) which is indicated in the current study by the decrease in clean swim speed, as was also shown in chapters 2 and 3. Fatigue has also been previously shown to hamper the sensorimotor system (Forestier and Nougier, 1998; Tripp, Yochem and Timothy, 2007) which affects the ability to maintain form and stability in movement patterns. It could be speculated that the alterations in the co-ordination patterns of the participants are a product of changes in the neuromuscular system. It has been reported that fatigue causes changes in the function of muscles that

normally perform the task resulting in changes in co-ordination of muscle activity (Forestier and Nougier, 1998) as other muscles attempt to assist with the task. These changes in muscle co-ordination and activity could result in a decreased power output which results in a decrease in clean swim speed, as shown in the current study. The significant decreases reported in the current study for clean swim speed and SL resulted in a significant decrease in SI. A decrease in SI indicates that there is a decrease in the stroke efficiency (Costill, et al., 1985), which could be as a result of the accumulation of fatigue which reduced the muscles ability to maintain force generating capacity (Gandevia, 2001). The alterations in the participants' stroke efficiency in the present study could be a direct result of fatigue. Fatigue has been defined as a failure to maintain power output (Edwards, 1983) which can be shown in the current study as a decrease in clean swim speed. Although no direct measure of change in force production of the participants was used in the current study the findings of chapter 2 and 3 support this line of discussion. In the previous chapters a decrease in force production of the arms and legs as measures using isometric contractions were reported following a 100 m breaststroke short course swim. Fatigue has also been previously shown to hamper the sensorimotor system (Forestier and Nougier, 1998; Tripp, Yochem and Timothy, 2007) which affects the ability to maintain form and stability in movement patterns. It has been previously speculated (chapter 3) that the alterations in the co-ordination patterns of the participants are a product of changes in the neuromuscular system. It has been reported that fatigue causes changes in the function of muscles that normally perform the task resulting in changes in co-ordination of muscle activity (Forestier and Nougier, 1998). These changes in muscle co-ordination and activity could result in a decreased power output which results in a decrease in clean swim speed, as shown in the current study. However the findings of the current study suggest that there were no alterations in the participant's co-ordination patterns from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m (Table 5.2 and 5.3). As there was no significant change in any of

the co-ordination phases of the stroke cycle, this means that hypothesis 4 can be rejected. A previous study (Oxford et al., 2010) has reported that a decrease in co-ordination phase one (CPhase1) may be a consequence of fatigue, which resulted in compensatory mechanisms of the neuromuscular system in an endeavour to maintain mechanical power output. In the present study the inter-length comparison showed that there was no change in the time spent in the propulsive or recovery phases of either the arms or the legs from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m (Table 5.3).

### 5.5.2 *sEMG Changes*

Neither the mean frequency of the power spectrum (MPF) nor the median frequency (MDF) showed any significant ( $p > 0.05$ ) changes from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m (Figure 5.1 and 5.2, Section 5.4.3, Chapter 5). This is consistent with previous research that had stated that MPF and MDF report similar findings (Bigland Ritchie 1981; Masuda *et al.* 1983, Brody *et al.* 1991). Therefore, as the MPF and MDF data show very similar results in the current study, only the MPF data will be discussed in keeping with previous swimming studies that have only reported MPF (Caty et al., 2007; Strin et al., 2011).

The MPF at the beginning and the end (MPF<sub>Beg</sub> and MPF<sub>End</sub>) of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of the swim showed no significant change for each of the twelve muscles observed in the current study. This does not support hypothesis 1 as it was expected that all muscles would report a decrease in frequency from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m. These results are not in accordance with the findings of Strin, et al., (2011) who reported a significant decrease of 20.5 – 24.6% decrease in MPF in the muscles pectoralis major, latissimus dorsi, biceps

brachii and triceps brachii during a 100 m maximal effort front crawl short course swim. The difference between the studies could have been due to the level of swimmers used and the fact that they used different strokes, which require different muscle activation patterns. The breaststroke stroke cycle is characterised by a glide phase of the legs and the arms which may be sufficient for the muscles to recover between strokes. The differences between studies may be due to greater reliance on the leg muscles, and thus the kick, in breaststroke, compared to the arms which are responsible for 80-90% of the propulsion generated in the stroke during front crawl swimming (Deschodt, 1999; Hollander, De Groot and Van Ingen Schenau, 1988). Another possible reason for the differences could be due to the methodologies used to calculate the fast Fourier transform (FFT), as previous works have not fully reported the treatment of the power spectrum, so it is difficult to know whether direct comparisons can be made.

A decrease in MPF is considered to be a result of a decrease in muscle fibre conduction velocity (Linssen et al., 1990; Lowery, Nolan and O' Malley, 2002; Strin et al., 2011) which has been shown to be associated with a decrease in pH (Allen, Lamb and Westerbald, 2008). Although pH was not measured in the current study, previous studies in breaststroke swimming have reported blood lactate concentrations around  $9.6 \pm 1.8 \text{ mmol l}^{-1}$  post swim (Thompson et al., 2003). High blood lactate concentrations imply a significant pH decrease during a swim (Strin et al., 2011). There are other factors besides muscle fibre conduction velocity decrease that could contribute to a decrease in MPF including: changes in the action potential (AP) duration (Dimitrova and Dimitrov, 2003; Merletti, Knaflits and De Luca, 1990); motor unit recruitment (Gazzoni, Farina and Merletti, 2004); and short-term motor unit synchronisation (Farina, Fosci and Merletti, 2002; Hermens et al., 1992). It has been suggested (Bigland-Ritchie and Woods, 1984; Dimitrova and Dimitrov, 2003; Gabriel

and Kamen, 2009) that both the peripheral muscle properties (i.e. motor unit size, changes in muscle fibre conduction velocity, AP shape and duration) and the motor control properties (i.e. changes in muscle recruitment patterns) need to be considered when interpreting changes in sEMG amplitude and in MPF, and not solely the physiological mechanisms related to fatigue (Strin et al., 2011). Although there was not simultaneous measurement of the amplitude changes from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m in the current study, amplitude was measured in chapter 4. The amplitude changes were calculated over three stroke cycles over four sections of the swim in the muscles pectoralis major, latissimus dorsi, vastus lateralis, vastus medialis, rectus femoris and biceps femoris. The findings of chapter 4 were that there was a significant decrease in the peak amplitude over the duration of the swim in the pectoralis muscle only. The findings of the current study and those of chapter 4 confirm that, in general, the muscles that were studied were recruited at a submaximal level and did not experience high levels of fatigue. Within the current study there were no significant changes in the co-ordination of the arms and legs however there is a tendency for participants to move towards greater overlap within the stroke cycle as reported in chapter 4.

All the muscles that were observed in the current study (Figure 5.11) would not be expected to fatigue to the same extent during the swim for two main reasons (Strin et al., 2011). Firstly, different durations of activation would be expected between the muscles as reported in Chapter 4; and secondly it depends on the role of the muscle in the execution of the stroke cycle. It was hypothesised (hypothesis 2) that the greatest fatigue would occur in the pectoralis major muscle of the upper body, due to its primary role in generating propulsion during the arm stroke (Nuber et al., 1986), and the vastus medialis and vastus lateralis, which are the key power producing muscles (Janshen, Mattes and Tidow, 2009; Ryan and Gregor, 1992; Wilson, Robertson and Stothard, 1988) that play an important role in



generating propulsion during the leg kick. The results of the current study mean that hypothesis 2 must be rejected. . The nature of the breaststroke technique requires that there is propulsion generated from both the arm and leg muscles this may explain the submaximal recruitment. Thus there is less reliance on a particular muscle group to generate propulsion when compared to the front crawl stroke (Strin, et al., 2011).

Overall there was no significant change in the MPF of the power producing muscles of the leg, vastus medialis and vastus lateralis. The leg muscles have previously been shown to be dominant in the development of propulsion during the breaststroke, with peak propulsion occurring during the leg kick phase of the stroke (Capitao et al., 2006; Craig, Termin and Pendergast, 2006; D'Acquisto and Costill, 1988; Yoshimurs et al., 2005). A decrease in muscle frequency has been shown to relate to a decrease in force production (Gerdle et al., 2000; Mac Issac, Parker and Scott, 2001; Potvin, 1997) which would go some way to explaining the decrease in clean swim speed from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m previously reported in breaststroke swimming (Thompson, et al 2000). Studies that have involved dynamic movements (Orloff, Craven and Gornick, 2011) have reported an increase in the MPF and have attributed the increase to compensatory mechanisms in an attempt to maintain total muscle power output due to an overall decrease in the quadriceps femoris muscle group (Orloff, Craven and Gornick, 2011). This was shown to increase regardless of whether fatigue occurs before (central fatigue) or after (peripheral fatigue) the neuromuscular junction (Boerio et al., 2005). The other dynamic stabiliser of the knee, the biceps femoris muscle, also showed no significant change in MPF over the duration of the swim. This is not consistent with previous studies that have looked at dynamic activities (Orloff, Craven and Gornick, 2011) and isometric studies (Boerio et al., 2005; Zory et al., 2005) as they reported

decreases in frequency. The differences may again be due to the fact that the leg muscles are working submaximally in the current where previous studies have used maximal activities.

There was no significant change in MPF of the pectoralis major and latissimus dorsi muscles over the duration of the swim. The pectoralis major and latissimus dorsi muscles play an important role in the production of propulsion in the arm stroke during extension, adduction and internal rotation of the glenohumeral joint. These findings are not consistent with the findings of Strin, et al., (2011) who reported an overall decrease in frequency of pectoralis major and latissimus dorsi during a 100 m front crawl swimming. This differences maybe due to the previously mentioned differences in the level of reliance of the pectoralis major and latissimus dorsi for the generation of propulsion due to the lesser role that the arms play in propulsion in breaststroke when compared to front crawl.

The muscles in the hip and trunk region showed a tendency ( $p = 0.05$ ) for a change in  $MPF_n$  between muscles, the gluteus maximus and the erector spinae reported a decrease in MPF over the duration of the swim, whereas rectus abdominal showed an increase in MPF. The trunk muscles play a very important role in the successful generation of propulsive force in the arms and legs in the aquatic environment (Prins, 2007). The trunk muscles provide a stable platform for the generation of propulsive forces (Prins, 2007). The decrease in the frequency of erector spinae and the gluteus maximus may suggest that these muscles are becoming fatigued. Such fatigue would result in a loss of stability of the trunk and a reduction in the ability to maintain propulsive force production of the arms and legs; also the distal muscles would lose the ability to generate force as there is no stable platform for the generation of force. It has previously been reported that there is a need for proximal stability for distal mobility (Kibler, 2001). The increase in the frequency of rectus abdominal muscle

may be a compensatory mechanism in an attempt to maintain hip, and lower lumbar trunk stability which provides a stable base for the muscles to act against. This loss of a stable base for the participants to generate force against could be one of the reasons why the swimmers experience a decrease in clean swim speed from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m.

Although in the current study there were no significant differences in the rates of change this is not what would have been expected. It would be expected that different muscles would experience differences in the rates of change in the MPF due to differences in muscle morphology (Strin et al., 2011). Changes in MPF occur more rapidly in muscles that contain a higher proportion of fast twitch fibres compared to those that contain a higher proportion of slow twitch fibres (Komi and Tesch, 1979; Larsson et al., 2003). The structure of the motor units will alter the muscular fibre conduction velocity, which can lead to a decrease in the MPF. Slower motor units will activate slow twitch muscle fibres at a lower frequency, due to lower muscle fibre conduction velocity, however these activations can be maintained for longer prior to fatigue. The fast motor units will activate fast twitch fibres at a higher frequency, due to faster muscle fibre conduction velocity, but these cannot be maintained for as long and there is drop out in the recruitment of motor units. Subsequently the decrease in muscle fibre conduction velocity is related to the proportion of fast twitch muscle fibres in the studied muscle (Strin et al., 2011). Consequently, as fatigue progresses, there is a decrease in the peak twitch tension which will result in an increase in contraction time as the faster muscles become fatigued, therefore the slower muscles will dominate the muscle force-time profile in trying to maintain the same impulse (Fuglevand, Macefield and Bigland-Ritchie, 1999; Thomas, Johansson and Bigland-Ritchie, 1991). This results in an overall decrease in the firing frequency of the muscle. A decrease in muscle firing frequency

has been related to a decrease in force production (Dideriksen, Farina and Enoka, 2011) however this cannot be supported from the findings of the current study.

## **5.6 Future Directions**

To fully understand the effects of fatigue during a 100 m breaststroke swim it would be of assistance to simultaneously analyse both the amplitude and frequency of sEMG (Strin *et al.* 2011), as a decrease in muscle amplitude has been shown to be associated with a decrease in frequency (De Luca, 1979). Such data needs to be viewed in conjunction with the kinematic data to investigate the changes in inter-muscular co-ordination and inter-muscular synchronisation. Unfortunately this was not possible in the current study due to limitations in the equipment that was available at the time the study was conducted.

Further research is required to substantiate the changes in the hip and trunk muscular activity and its relationship to a swimmer's ability to maintain a stable base for the generation of propulsive forces. This could be a key area for improvement in performance times. The maintenance of a stable base should allow the swimmer to maintain optimum levels of power production from the arms and legs. If the hips, trunk and lower lumbar region can remain stable for longer it may mean that swimmers are able to maintain optimum combinations of stroke length and stroke rate for longer time periods, which would increase overall mean clean swim speed. This needs to be investigated across all the swimming strokes, not just the breaststroke technique.

The current study does not consider the differences in asymmetry between the dominant and non-dominant side of the body, especially with regards to the hip and trunk

musculature. This needs to be investigated to give a better understanding of the stability of the hips, trunk and lower lumbar region and swimming performance. An investigation that could also calculate the intra-cyclic velocity fluctuations would be extremely useful if combined with both EMG amplitude and frequency data as this would allow direct simultaneous assessment of changes in muscle activity and swimming speed, especially during the propulsive phases of the legs and arms of the stroke cycle. This could possibly be achieved using a 3D kinematic investigation with time synchronised sEMG analysis.

## **5.7 Conclusion**

This study has investigated the changes in the frequency spectrum MDF and MPF over the 1<sup>st</sup> and 2<sup>nd</sup> 50 m of a 100 m maximal breaststroke swim. The investigation involved twelve muscles of which four were in the lower limb, three in the trunk region and five in the upper body. The main findings of the study were that there was no significant change in the MPF when normalised from the beginning to the end of the 1<sup>st</sup> and 2<sup>nd</sup> 50 m respectively between muscles or between the beginning of the 1<sup>st</sup> and the end of the 2<sup>nd</sup> 50 m. The investigation also investigated the changes in kinematics from the 1<sup>st</sup> to the 2<sup>nd</sup> 50 m with significant decreases evidenced in clean swim speed, stroke length and stroke index. There were no significant changes in the co-ordination of the arms and legs, or in the time spent in each of the arm and leg phases. The findings from this study suggest that there was a decrease in the participant's stroke efficiency but that this cannot be explained by changes in the frequency spectrum of the surface EMG.

**CHAPTER 6**  
**GENERAL CONCLUSION**

## CHAPTER 6

### 6.0 General Conclusion:

#### 6.1 Summary:

The overall aims of the thesis were to evaluate the biomechanical and physiological changes that occur during a 100 m breaststroke swim. Through the analysis of the kinematics, kinetics and physiological parameters this thesis has presented new and in-depth contributions to research in swimming biomechanics, especially with respect to breaststroke swimming. There is now a greater understanding of the co-ordination, intra-cyclic velocity variations, physiological and electromyography changes that take place during a 100 m breaststroke swim. The following is a summary of the main findings of the thesis:

1. The findings of the thesis demonstrate that as participants progress through a 100 m breaststroke swim there is a significant decrease in clean swim speed ( $p < 0.05$ ) (Chapter 2, 3, 4 and 5). The decrease in clean swim speed can be attributed to a significant decrease in stroke length (Chapter 2, 3), with no significant changes in stroke rate observed over the duration of the swim. The significant decrease in clean swim speed and stroke length resulted in a significant decrease in stroke efficiency (chapter 2, 3 and 5). The decrease in stroke efficiency was not related to changes in activity of the 12 muscles, as demonstrated by EMG analysis (chapter 5).

2. Anthropometric characteristics of males and females such as size and surface area of propulsive segments are factors that have an impact on performance (Chapter 2). Males had

significantly greater clean swim speeds compared to females (chapter 2). The greater clean swim speeds can, in part, be explained by the males being significantly taller and having significantly longer segments lengths for the hand and the forearm (Chapter 2). The increased propelling surface that is available to the male swimmers potentially allows them to generate more propulsion than females.

3. Arm-leg co-ordination was investigated by the assessment of the time gaps that quantify the continuity between the movements of the arms and the legs. It was shown that small changes in the time gaps between leg and arm movements (Chapter 2 and 3) could reveal great disturbance to motor organisation. There does not appear to be an ideal co-ordination pattern as different profiles of co-ordination appear to relate to different combinations of stroke rate and stroke length (Chapter 2 and 3) and therefore alterations in strokes efficiency (Chapter 2 and 3).

4. The dry land strength capacity of the upper and lower body significantly decreases following a 100 m breaststroke swim (Chapter 2, 3), but does not appear to be related to the changes in net propulsion of the arms and legs (Chapter 3).

5. The analysis of EMG amplitude as a percentage of  $\text{Peak}_{Task}$  for rectus femoris, biceps femoris, vastus lateralis and vastus medialis of the lower body and petoralis major and latissimus dorsi of the upper body showed significant changes in magnitude over four 25 m sections of the 100 m breaststroke swim (Chapter 4). There were significant changes in the active and non-active phases of the muscles from the 1<sup>st</sup> 25 m to the 4<sup>th</sup> 25 m of the 100 m swim. There was no significant relationship between muscle amplitude and arm-leg co-ordination (Chapter 4).



6. Frequency spectrum analysis identified that the major upper and lower body muscles used in breaststroke swimming are activated at submaximal levels, such that there are no significant changes in the median power frequency or mean power frequency over the duration of a 100 m breaststroke swim.

Therefore, the results of the current thesis indicate that there are two distinct co-ordination patterns utilised by the participants during a 100 m breaststroke swim. These are the *Glide*, co-ordination pattern, which is characterised by a glide phase following the end of the arm recovery phase prior to the start of the leg kick, and the *Overlap* co-ordination pattern, which is characterised by the initiation of the leg kick before the completion of the arm recovery phase (Maglischo, 2003; Seifert, et al., 2011). This finding is not in line with previous research (Chollet, et al., 2004; Leblanc, et al., 2005; Seifert & Chollet, 2005) as these studies used discontinuous graded protocols of 25 m. These previous studies have assumed that the different co-ordination patterns adopted by swimmers correspond to swimming speeds used during 50-200 m swims.

The findings of the current thesis confirm that there are differences between the sexes with regards their ability to generate force, as shown by differences in hand and isometric leg strength. There were also differences between the sexes regards height and also segment lengths. Males reported significantly greater stature and hand and forearm segments which resulted in significantly greater stroke lengths greater clean swim speeds.

The findings of the thesis demonstrate that as participants progress through a 100 m breaststroke swim there is a significant decrease in clean swim speed ( $p < 0.05$ ; Table 2.3, Section 2.4 ; Table 3.2, Section 3.4; Table 5.1, Section 5.4). The decrease in clean swim speed can be attributed to a significant decrease in stroke length ( $p < 0.05$ ; Tables 2.3, 3.2 and 5.1) however there is no subsequent decrease in stroke rate ( $p > 0.05$ ; Tables 2.3, 3.2 and 5.1) over the duration of the swim. This decrease in stroke length is consistent with previous studies (Takagi, et al., 2004; Thompson, et al., 2000). The study of stroke length and stroke rate does not give an insight into the swimmers underlying ability to generate propulsive force or the amount of the resistance force they experience during the recovery phases of the stroke. Chapter 3 used measurements of instantaneous velocity during a swim to gain insight into the fluctuation in propulsive and resistance forces underlying swimming performance during a 100m breaststroke swim. The breaststroke swimming stroke involves large fluctuations in linear speed, as shown in chapter 3. The results show that as the swimmers progressed through the swim there was a decrease in the peak velocity during the arm and leg phases (Table 3.3, Section 3.4.1) and a significant decrease in the minimum velocity recorded during the recovery phase ( $p < 0.05$ ; Table 3.3, Section 3.4.1). There was a significant decrease in the isometric hand and leg strength from pre to post swim. It has been previously reported that a swimmer's speed is directly affected by their ability to generate a mechanical output (Toussaint & Truijens, 2005). The results of the thesis suggest that during the 100 m swim the participants are becoming fatigued, as fatigue has been defined as a decrease in the ability to maintain effective mechanical output (Beelen and Sargent 1991). The suggestion of fatigue was further supported by the finding of the surface electromyography studies (Chapter 4 and 5) where there was a significant decrease in the active phase of the leg muscles ( $p = 0.03$ ; Figure 4.6, Section 4.4.2) as the participants progressed through the 100 m swim.

The findings of the current thesis can only infer that, as a consequence fatigue process that during a maximal 100 m breaststroke swim, participants used compensatory mechanisms and strategies in an attempt to maintain clean swim speed; these mechanisms and strategies resulted in alterations in the combination of stroke rate and stroke length, altered intra muscular co-ordination and changes in the arm-leg co-ordination within the stroke cycle.

## **6.2 Future Directions**

The work presented in the thesis has examined the biomechanical and physiological changes that take place during a 100 m breaststroke swim. As described in section 6.1 we have presented some novel findings that add to the body of research in breaststroke swimming. However, in light of these findings a number of additional questions have been raised which warrant further examination. They are as follows:

1. We showed that there are no relationships between dry land isometric strength and intra-cyclic velocity variations (Chapter 3). Further work should consider different possible mechanics to test dynamic strength in the aquatic environment and relate these to intra-cyclic velocity variations. Further research should also consider whether long term changes in the ability to generate force of the arms and legs of swimmers are related to increases or decreases in arm and leg propulsive forces and how these change during a swim.
2. We used competent breaststroke swimmers throughout the thesis however further research would benefit from using Elite level breaststroke swimmers. Previous

research has already shown that there are differences in arm-leg co-ordination in breaststroke swimmers with respect to skill level.

3. In the current study we investigated changes in co-ordination in male and female swimmers (Chapter 2) however there were no female subjects included in the samples for the intra-cyclic velocity variation studies or the EMG studies. Further research would also need to consider sex differences as it has previously been shown that there are sex differences in arm-leg co-ordination in breaststroke.
4. We evaluated the active and non-active phases of muscle activity during a 100 m breaststroke swim. However, these findings were not related to the inter muscle co-ordination and co-activation mechanics of the muscles. Further research should focus on activation vs. non-activation phase for agonist and antagonist muscles, for principal muscles and periphery muscles used during breaststroke swimming. This would increase the understanding of muscle co-ordination with in breaststroke swimming.
5. We investigated the changes in co-ordination of the arms and legs and the muscle activity of twelve major muscles used in breaststroke swimming. Further research is required to synchronise the co-ordination of the arms and legs with the EMG analysis. This would provide a more detailed analysis of changes that occur in arm and leg co-ordination and the subsequent activations of the prime and peripheral muscle used during a breaststroke stroke cycle.
6. In the current study we only investigated the muscle activity of twelve muscles from the right hand side of the body. Further research is required to analyse

potential bilateral differences in muscle activity. This would provide insight in to whether any neuromuscular changes are equal on each side of the body. Further to this it would also be valuable to analyse potential strength differences between both sides of the body to see if that relates to differences in muscle activity.

7. We investigated muscle fatigue via the analysis of spectrum analysis, however further research is required to use more sophisticated EMG analysis techniques such as wavelet analysis to provide more in depth analysis. Wavelet analysis would enable the analysis of co-activation of primary muscles and potentially relate these changes with arm –leg co-ordination.

## **6.3 Limitations**

### *6.3.1 General limitations*

The general limitations of the thesis are that there was a dearth of experienced male and female breaststroke swimmers that were available to participate in the research. The limited subject population did not allow for the recruitment of sufficient females to be included in the thesis findings thus there data was removed from the latter thesis chapters. The removal of the female participants reduced the total number of participants and therefore reduced the statistical power of the findings of studies 2, 3 and 4 (Chapters 3, 4 and 5).

### 6.3.2 *Experimental Limitations*

Although great care and attention has gone into this research, as always there are some experimental limitations that have been observed. They are as follows:

1. Throughout the thesis the methods utilised 2D video analysis techniques which recorded at 50 Hz. A limitation of the low recording rate is that it makes analysis of dynamic movements less accurate. When determining whether a participant utilised the glide, continuous or overlap techniques there were very large movements of limbs that occurred within the 0.02 s time frame. This could make the difference between a participant being categorised as a glide swimmer or an overlap swimmer. However, this limitation does not alter the observed trends in the data that demonstrated a reduction in glide phase or increase in overlap as the 100 m swim progressed. It would be recommended that higher frame rates should be used in the future. The higher frame rate would have increased the accuracy of the intra-cyclic velocity calculations from the greater trochanter (Chapter 3). A high frame rate would have allowed closer comparisons to the fixed methods of determining intra-cyclic velocity variations.
2. During the experimental trials the candidate was unable to record the swimmer from both sides of the body therefore it was assumed that each participant had equal symmetry on both sides of the body. This is a limitation in the analysis of the co-ordination of the participants. However, there is limited research to suggest there are asymmetrical differences. Thus the findings are in line with previous research in breaststroke swimming.

3. One of the limitations of the current thesis was that the sEMG was not synchronised with the video analysis such that changes in co-ordination between movements of the arms and legs could not be attributed to changes in muscle co-ordination and changes in muscle activities. One of the other limitations with using sEMG is that the participant is tethered to a rod that holds the EMG clear of the water. This process interferes with the turning ability of the participants and influences their turning techniques, thus reducing overall performance time. However, even the methods used in the current study are in line with previous research into surface EMG in swimming.

#### **6.4 Practical implications**

The improvement of biomechanical methods such as surface EMG and video analysis techniques helps to grasp a better understanding of the breaststroke. The finding of the current thesis shows that there are large intra-individual variations observed between fresh and fatigued in kinematics, kinetics and EMG. Consequently, individual biomechanical approaches could be a useful tool in evaluating swimmers performance and in the adoption of specific training process. Coaches would be advised to look at the intra-cyclic velocity variations, co-ordinations strategies and body characteristics before recommending specific drills and training interventions with their swimmers.

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